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NORTH CAROLINA
Medical Journal

July, 1961
Vol. 22 No. 7

IN THIS ISSUE:

Annual Report of the State Board of Health

J. W. R. Norton, M.D.

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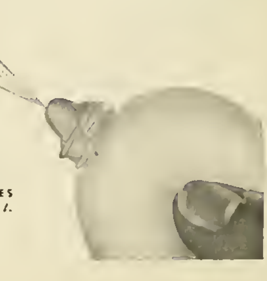

NORTH CAROLINA MEDICAL JOURNAL

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THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

VOLUME 22 JULY, 1961 No. 7

Annual Report of the North Carolina State Board of Health, 1961

J. W. R. NORTON, M.D., M.P.H., F.A.C.P.*
RALEIGH

The customary detailed annual report of activities in state and local public health work is provided for the Medical Society. Instead of summarizing, we felt it would be more interesting, at this time, to touch on some of the high lights and then to mention a few problems of general mutual interest. We take up the high lights by our divisional organization.

Laboratory Division

LYNN G. MADDRY, Ph.D., Acting Director

Among the achievements for 1960 were the provision of services for rapid identification of beta hemolytic streptococci group A by the fluorescent antibody procedure and the complement fixation tests for fungus diseases.

A serious problem is our inability to recruit and retain properly trained personnel because of the low starting salary range. The clerical staff is burdened beyond the point of being able to take care of the increased volume of work, and it is necessary that the technical staff perform some of these duties. This in turn reduces the number of specimens that can be examined.

It was disappointing that, owing to limitation of state funds, we are unable to meet the needs of the people in North Carolina in the field of cancer cytology, virology, and environmental health.

Oral Hygiene Division

E. A. PEARSON, D.D.S., Director

This marks the thirtieth anniversary of our Oral Hygiene Division. The main emphasis is still on providing for children in the elementary grades a program of dental health education which includes, insofar as funds and staff will permit, the following: classroom instruction and dental inspection for all; dental correction for the underprivileged; and referrals of other children to private practitioners.

Of special significance in the progress of the program during the past year are the developments in the areas of recruitment, research, and fluoridation.

The rapid turnover of staff dentists makes recruitment a constant problem. New approaches in the form of a recruitment brochure mailed to senior dental students of eastern and southern dental schools, and visits to many of these schools by the Division Director, have brought about a dramatic change: We now have more applicants than openings. Eight new dentists will begin work in July, thereby enabling us to provide dental health programs in many more counties than heretofore.

The Division's participation with the U.S.P.H.S. in the state-wide research project, "The Natural History of Dental Diseases in North Carolina," is progressing well. Dental examinations have been completed for 20 per cent of the households in the total sample. The consummation of this project will provide, for the first time, prevalence rates of dental diseases for a state

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Read before the Conjoint Session, Medical Society of the State of North Carolina and the North Carolina State Board of Health, Asheville, May 10, 1961.

*Secretary-Treasurer, North Carolina State Board of Health, Raleigh.

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population. Such information will be invaluable in helping us to define our problems and to measure progress.

Recognizing that fluoridation is the most effective method of preventing tooth decay, we are glad to report progress in the fluoridation of municipal water supplies. Since our last meeting three cities have adopted this public health measure, making a total of 34 North Carolina municipalities now adjusting fluorides upward to the optimum amount (one part per million) for dental development and health. The combined population of these cities is 1,036,507 and represents 55 per cent of the urban population. Fluorides occur naturally in amounts of 0.7 to 1.2 ppm, in the water supplies of 17 other North Carolina municipalities having a total population of 35,569. Five also have natural fluorides in amounts beyond 1.2 ppm: Winton (1.4), Williamston (1.5), Wentworth (1.8), Rich Square (2.1), and Windsor (3.0).

Personal Health Division

JAMES F. DONNELLY, M.D., Director

1. Responsibility for setting standards and licensing of nursing homes was transferred from the Medical Care Commission to the North Carolina State Board of Health on March 17, 1961. The school health program was also transferred from the Division of Local Health to Personal Health on March 16, 1961.

2. Two new cancer detection clinics were added, to bring to 19 the detection clinics now functioning. An additional 4,000 Papanicolaou smears were examined by the Laboratory Division, bringing the total for 1960 to 35,514.

3. Physiotherapists were added to the heart programs in three counties. We now have four physical therapists on the crippled children's and chronic disease programs.

4. A stroke rehabilitation program emphasizing home nursing care was initiated in five counties.

5. A survey program for diabetes was initiated in 20 counties, with particular stress on the nutrition of the pre-diabetic patient discovered in the survey.

6. Nutritional programs in industry were expanded, and there was marked expansion in consultation and review of architectural plans for institutional kitchens.

7. The Crippled Children's Section instituted changes in reporting statistics and recording financial and medical information on crippled children's cases, to provide a clearer basis for evaluation of these services. This Section also has raised the age limitation from 15 to 21 for presentation to rheumatic fever and speech and hearing clinics, and it has assumed clinic observation, preoperative and postoperative, of patients having congenital and other types of heart disease.

8. The number of midwife licenses continued to fall, reaching a new low of 294.

9. Follow-up study of prematurely born infants in Wake County was completed, and the results have been analyzed and reported.

Sanitary Engineering Division

J. M. JARRETT, B.S., Director

Two reports of statewide significance were printed and distributed. One summarized the results of an 18-month study of background radiation in surface streams used as sources of public water supplies. One hundred thirty-five sampling stations were established on major streams of the state for periodic checking.

The second report shows results of the "Air Pollution Study of North Carolina" made in 1958-1959 as a special project conducted in cooperation with the U. S. Public Health Service. The survey indicates the need for control activities throughout the state. Additional funds from the legislature are being requested to enable us to develop this program and provide protection against such unfortunate hazards as have developed in Donora, Pennsylvania; Los Angeles, California; London, England; and to lesser degrees elsewhere.

Regulations for sanitary control of mechanical vending machines were adopted, but operation has been delayed until the necessary preliminary education program can be carried out.

In addition to continuing our participation in the air radiation surveillance network, North Carolina was added to the Public Health Service surveillance network for radioactivity in fluid milk supplies when Charlotte was added to the list. Samples are examined at the Public Health Service Radiation Laboratory, Montgomery, Ala.

There are presently 650 public water supply systems under supervision, with 45 new systems added during 1960. Costs of water supply improvements last year amounted to $3,609,372.00.

This Division and local staffs now have 22 engineers and 248 sanitarians providing environmental health protection.

Local Health Division

Robert D. Higgins, M.D., Director

Administration

One of the primary objectives of this Division has been to consolidate wherever and whenever feasible the smaller single county health units into district units of two or more counties. Some of our single county units could provide only a part-time health program, but by joining with one or more neighboring counties, they could establish a full-time program under the direction of a qualified full-time director at a salary commensurate with his duties and with improved services in each participating county in the district. Sixty-nine departments serve the 100 counties, since 18 district departments serve 49 counties.

Through an incentive (50 per cent of cost up to $1,000 per county) offered by the State Board of Health to counties which are under, or those which do come under it, the Local Governmental Retirement Plan has grown rapidly, and 61 of the 100 counties now provide their local health employees with this coverage.

Public health nursing

The public health nurses have made progress in their goal to provide improved nursing care to patients referred by physicians in private practice or in local, state, and regional hospitals. The objectives of the service are to permit early discharge from the hospital, assist family members in nursing techniques, and promote rehabilitation and restoration.

1. Plans have been developed whereby physicians in the Veterans Administration Hospitals refer selected patients as discharged to local health departments for nursing service. The public health nurse makes one visit to the patient, reminding him of the need for continuing local medical care, and returns only if the patient is under the care of his local physician. This service is provided in 96 of our 100 counties.

2. Additional health departments are now providing public health nursing services to the patients discharged from our state and Veterans Administration psychiatric hospitals and to their families (20 counties). The state hospital medical staffs are enthusiastic about the service, and only the lack of hospital and public health personnel prevents extension of this valuable service which has decreased the number of readmissions in these counties.

3. The referrals from North Carolina Memorial Hospital have increased in number and urgency. Throughout the country this is regarded as an outstanding example of an efficient, well organized plan for referral between a teaching hospital and local nursing resources.

4. Demonstrations of how limited nursing service to the chronically ill may be provided by busy general public health nurses are encouraged, and have been started in Halifax, Alamance, New Hanover, Person, Mecklenburg, Guilford, Orange, Chatham, and Lee health departments. These have had the enthusiastic support of the local medical societies and the State Society Committee On Chronic Illness.

The chronically ill and aged in nursing homes and boarding homes have received improved services through direct consultation and in-service education for the staffs of these homes.

Health education

This Section promoted a three-day Workshop on Communications, in Raleigh, for 90 professional employees for the improvement of intra-agency communication. The staff consisted of people with special skills in the fields of the behavioral sciences, business and industrial personnel management, and public relations, as well as the disciplines of

Health, Perinatal Mortality, Child Health, and Anesthesia.

Home and farm accident prevention

In this important field, the Division:

1. Assisted in establishing two additional Poison Information Centers (Charlotte and Mount Airy) in addition to the three already in operation (Durham, Wilmington, and Jacksonville).
2. Followed up studies of drownings, accidental deaths among school-age children, and mortality due to fires and explosions.
3. Continued cooperation with Rural Safety Councils, Teachers' Workshops, and Off-the-Job Accident Prevention Councils.
4. Promoted staff education and in-service training of State and local cooperating agencies.

An increase of infectious syphilis was reported, with a total of 13,052 cases of venereal disease and 854,433 serologic tests by state, county and other approved laboratories.

Administrative Services Division

CHARLES L. HARPER, M.S.P.H. Director

A greater than usual amount of time, planning, and effort was put into preparing budgets for presentation to the Advisory Budget Commission and the Joint Appropriations Committee of the 1961 General Assembly, particularly by Dr. Jacob Koomen and Mr. Charles Harper. Recommendations made by the Commission and the serious consideration now being given by the Committee are encouraging.

A Travel Manual was adopted during the year to consolidate and summarize necessary policies and procedures for official travel of departmental employees.

A carefully prepared Biennial Report was published.

The Film Library supplied well over 17,000 requests, which was an increase of nearly 2,000 over the previous year.

An editorial board for the Health Bulletin was named and several changes, which we hope will be improvements, were made in this publication.

General Comment

Many items of continuing conjoint concern have called for attention since our last meeting. These include:

1. Fragmentation of medical and health services with favoritism toward non-medical leadership, as illustrated by the transfer of hospitalization funds of the Medical Care Commission to the Welfare Department by the present General Assembly, with pay-off in a higher ratio of federal support. This subtle technique will probably not stop with one quick success toward welfare control.
2. Medical plans for aged—national and state legislation, and implementation.
3. Two White House Conferences—with publicity designed to discredit the medical profession by certain highly aggressive groups.
4. National Committee Study Reports with recommendations proposing sweeping changes—Bayne-Jones, Cohen, Bane, and Jones, for example.
5. Continuing activities by a few physicians, particularly some in private practice, to alienate other medical doctors rather than to unify medical leadership and support.
6. A growing misinformed segment of the public which increasingly interprets medical leadership and guidance as resented medical dictation—and the general public will determine certain trends with or without appropriate information or medical leadership and guidance.
7. Voluntary prepayment plans. We say the success of voluntary prepayment plans for hospitalization and medical care lessens pressures toward compulsory systems, but we hasten the pricing of these voluntary plans out of the market by excessive x-ray and laboratory tests, and by excessive hospitalization for convenience. A few bright areas in the other direction are encouraging.
8. Why in the midst of plenty, health, and good living is there growing de-

pendence, misery, hunger, and destitution? Are palliatives and handouts the best course toward alleviation and improvement? What should or can the medical profession do, which we are not doing, to correct this socio-economic problem? Here again, our recognized understanding and appreciation are basic for our communication with those whom we serve.

The Management of Infants Suspected of Having Congenital Heart Disease

MADISON S. SPACH, M.D.
AND
JEROME S. HARRIS, M.D.
DURHAM

The management of congenital heart disease in infancy poses a dilemma. We know that one-half per cent or more of all children are born with some sort of cardiac malformation. Many of these malformations, which cause death in early infancy, could be diagnosed by modern means and corrected by advanced techniques in cardiac surgery. On the other hand, a great many murmurs can be heard in apparently normal infants. Many will disappear by the end of the first year, while others, though persistent, indicate only a minimal alteration compatible with normal life. It is therefore imperative that we chart our course between overemphasis on the investigation of infants with murmurs, on the one hand, and overcomplacence or pessimism regarding children with actual signs of congenital heart disease, neither assuming that they will grow out of it nor, if severe, thinking that nothing can be done about it.

Transient and Functional Murmurs

In the first few days of life murmurs can probably be heard in 5 per cent or more of children, particularly under the optimal conditions of a sleeping infant and a quiet room. These murmurs are usually systolic in time but not very loud, ranging between 1 and 2 plus in a total loudness scale of 4 plus. Many may result from an incomplete change from fetal to postnatal circulation, and will disappear within a week or two. It is known, for example, that both the ductus arteriosus and the foramen ovale may stay open for a variable period after birth in the normal neonate.

In addition to these very transient murmurs, there are functional murmurs which are often maximal in the third to fourth left interspace and may simulate those of interventricular defects. Many of these murmurs, however, will disappear by the end of the first year of life. The child with this type of murmur is asymptomatic, has normal heart sounds and a normal-sized heart, and shows a gradual diminution in the loudness of the murmur at successive monthly examinations. A normal electrocardiogram gives confirmatory evidence. Because soft to moderately intense systolic murmurs frequently disappear in the first six months of life, it is highly debatable whether the physician should tell the parents of this finding unless the child shows some symptoms or signs referable to the cardiovascular system, or unless the murmur increases in intensity at successive examinations. More harm than good may be done in such instances by creating suspicion in the parents that their child has a damaged heart.

Loud or Persistent Murmurs Without Symptoms

A second group of children shows a murmur which either is quite loud and associated with a thrill, or which increases with time or persists beyond the first year of life. The majority of these are probably associated with some definite change in the structure of the heart or circulation. Never-

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From the Department of Pediatrics, Duke University School of Medicine, Durham, North Carolina.

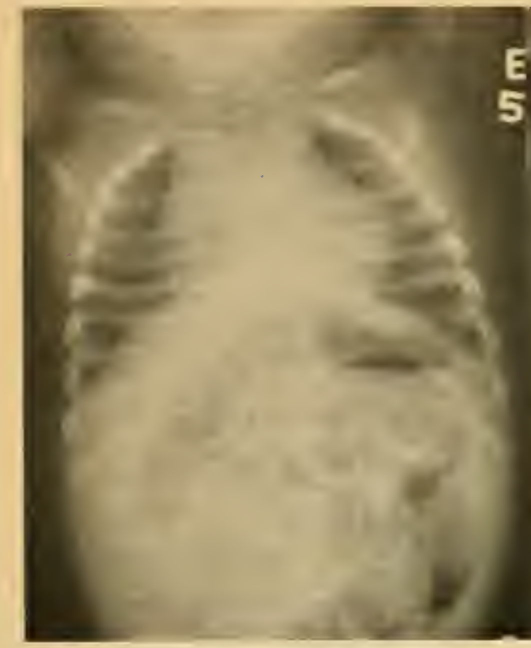

Fig. 1. Roentgenogram made during full expiration, showing "apparent" cardiomegaly.

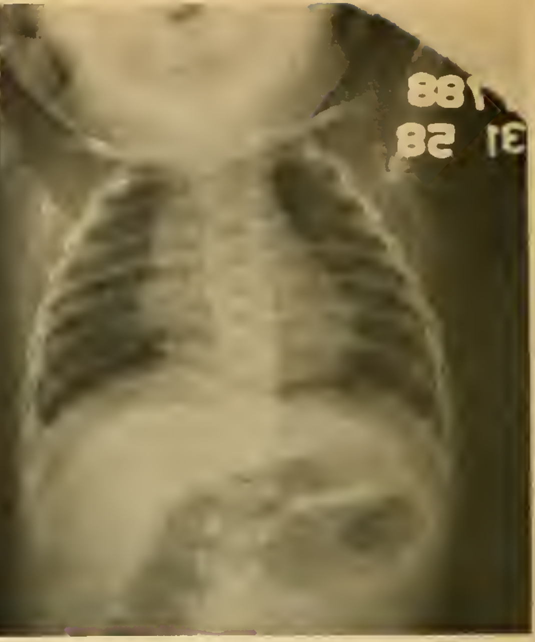

Fig. 2. Cardiac shadow appears within normal limits (full inspiratory film).

theless, the abnormality may be insufficient to cause any symptoms, signs or laboratory evidence (x-ray and electrocardiogram) of disease.

In contrast to true functional murmurs, these can be labeled organic but clinically insignificant. They may remain so throughout life or may cause difficulties later in periods of pathologic (that is, disease) or physiologic stress (for example, adolescence, child-bearing, and so forth). These children should be followed every three to four months during infancy and at increasing intervals thereafter by careful inquiry into the presence of cardiovascular symptoms and by meticulous physical examinations. At somewhat longer intervals, roentgenograms and electrocardiograms can be taken to determine whether the condition is still clinically insignificant. In our present state of knowledge there is no actual need for any further diagnostic tests during infancy in this group, since even if minor deviations (such as a very mild pulmonary stenosis or a mild interventricular septal defect) were to be found, surgery would not be indicated.

A word of caution should be interjected here concerning the interpretation of roentgenograms and electrocardiograms in infancy. If films are taken during expiration, the size of the heart may be greatly exaggerated and suggest a severe cardiac anomaly. Additional films taken during inspiration may reveal a completely normal heart (figs. 1 and 2). During early infancy, the electrocardiogram *normally* shows signs pointing to right ventricular dominance. The electrocardiogram, therefore, should be compared with controls of the same age rather than with the normal adult electrocardiogram.

Since it will be necessary to follow these children for a long period, it seems wisest to inform the parents of the murmur but at the same time to reassure them by pointing out that the condition has not caused any evidence of strain on the heart. If there is undue parental anxiety, the patient can be referred at this time to a cardiac center for confirmation of the physician's diagnosis. This will be reassuring and allow the cardiac center to establish a baseline against which comparison of the child's progress may be made.

Cardiovascular Symptoms With or Without Murmurs

The last group includes those infants who show evidences of cardiovascular difficulty with or without the presence of murmurs. These difficulties may be manifested by alterations in the cardiac rhythm, such as paroxysmal tachycardia, evidences of stress in cardiac enlargement or electrocardiographic abnormalities, cyanosis, cardiac failure or symptoms which at first glance may not suggest heart trouble. These include a low birth weight, poor weight gain, feeding difficulties, poor sucking, easy fatigability, excessive perspiration, increased susceptibility to respiratory infections with recurrent bouts of apparent pneumonia, rapid breathing, respiratory distress, or an acute shock-like state. In the infant, cardiac failure is almost always clinically right-sided regardless of the site of the lesion. Furthermore, edema is uncommon. More characteristic is enlargement of the liver, with evidence of pain or tenderness.

Frequently, the onset of frank cardiac failure in infants with large left-to-right shunts is preceded by bouts of apparent pneumonia, which are probably episodes of mild failure precipitated by respiratory infections. X-ray may show infiltrations which are largely the result of the very marked increased vascularity caused by the left-to-right shunt, but are interpreted as true pneumonias. Repeated bouts of pneumonia in a child who is frail and not thriving well should suggest the possibility of an acyanotic congenital heart disease with a large shunt.

This group of infants should be referred to a cardiac center for evaluation. Although many will not require diagnostic studies other than routine roentgenograms and electrocardiograms nor need surgery until much later, their course may suddenly change and require rapid surgical intervention. The mortality of congenital heart disease is greatest during the period of infancy, and particularly during the first six months of life. Many of these infants, if diagnosed properly, can be saved by prompt surgery. Indeed it has been said that one third of those infants who die of congenital heart disease during the first month of life could be benefited by surgery. The proportion increases with age.

Left-to-right shunts

One of the most potentially salvageable groups is comprised of infants who have large left-to-right shunts. Characteristically, these infants tend to do well clinically during the first few months of life, but are prone to develop congestive heart failure between the fourth and seventh months, if they are going to get into difficulty prior to later childhood. Such infants require close observation during this critical period of the mid-first year.

The main defects causing such difficulties are (1) interventricular septal defects, (2) patent ductus arteriosus, and (3) certain types of interatrial septal defects which lie close to and may be associated with deformities of the mitral and tricuspid valves (the ostium primum type of interatrial defect). It is interesting that the more familiar patent foramen ovale and interatrial septal defect usually do not cause clinical difficulty during infancy.

Diagnostic techniques

These three types of patients are usually acyanotic and have large hearts, increased pulmonary vascularity, and systolic murmurs. Since the patent ductus may not develop the diastolic component until late infancy or early childhood, these defects are often clinically indistinguishable. The electrocardiogram frequently is of value in separating the ostium primum defect from the patent ductus and the interventricular septal defects. A definitive diagnosis between the latter two anomalies frequently can be made only by means of cardiac catheterization or retrograde aortography. Both techniques can be done at any age.

Catheterization in infants does not differ in principle from that familiar in adults. Not infrequently the presence of a patent ductus or an interventricular septal defect

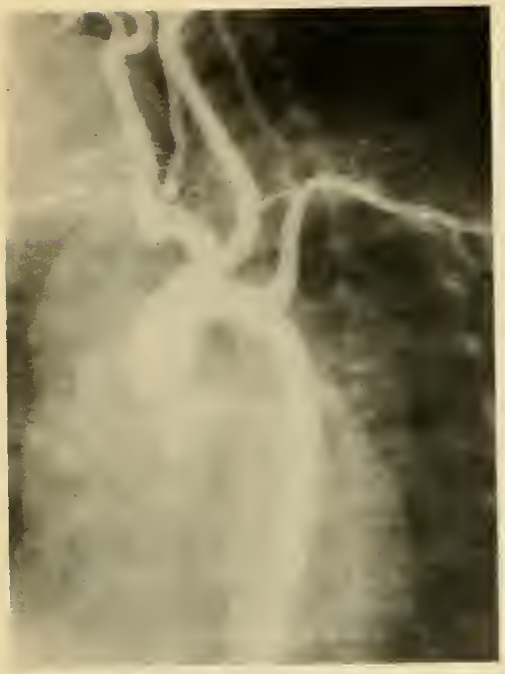

Fig. 3. Retrograde aortogram revealing normal aortic arch. The sinuses of Valsalva and coronary arteries are also outlined in this study.

can be demonstrated conclusively by passage of the catheter through the defect (for example, from the pulmonary artery directly into the descending aorta in the case of a patent ductus).

Retrograde aortography in infants is performed by the injection of radio-opaque material through a needle into the brachial artery, utilizing a special high pressure syringe. This forces the "dye" to pass in a retrograde fashion against the blood current to fill the aortic arch and its branches. If the pulmonary vascular system fills simultaneously with the aorta, the diagnosis of a connection between the aorta and pulmonary system (such as a patent ductus) is demonstrated. Figure 3 shows a normal aortic arch as outlined at retrograde aortography, while figure 4 shows the simultaneous filling of both the aortic and pulmonary systems in the case of a patent ductus arteriosus. Incidentally, the location of the ductus may be marked by an outpouching of the aorta at that point. This technique, as might be expected, is also very useful in the diagnosis of aortic arch anomalies and coarctations.

Other acyanotic conditions which are prone to cause difficulties during infancy are (1) coarctations (particularly when situated proximal to the insertion of the ductus), (2) fibroelastosis, (3) myocarditis, (4) disturbances of rhythm such as paroxysmal auricular tachycardia, and (5) a group of miscellaneous and bizarre abnormalities. The coarctations may be diagnosed if the physician attempts to palpate the femoral arteries. This is simpler and more effective than obtaining a blood presure. Occasionally, normal pulsations may be felt in the femoral arteries, but in this event the lower extremities will be cyanotic since the blood supply is partly from a patent ductus with reversed flow. Paroxysmal tachycardia is easily diagnosed by auscultation during an attack, and may be confirmed by an electrocardiogram. The fibroelastosis and myocarditis groups are more difficult and are essentially diagnoses of exclusion after all other defects have been eliminated. Murmurs may or may not be present, but cardiac enlargement is usually fairly marked. These, together with cases of paroxysmal tachycardia, are treated best by digitalization.

Malformations with Cyanosis

Finally there is the cyanotic group of in-

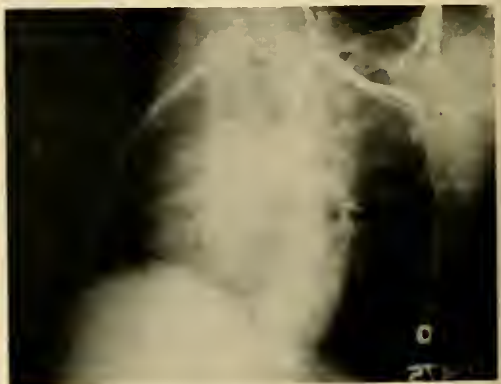

Fig. 4. Retrograde aortogram showing simultaneous filling of the aorta and pulmonary vessels, through a patent ductus arteriosus.

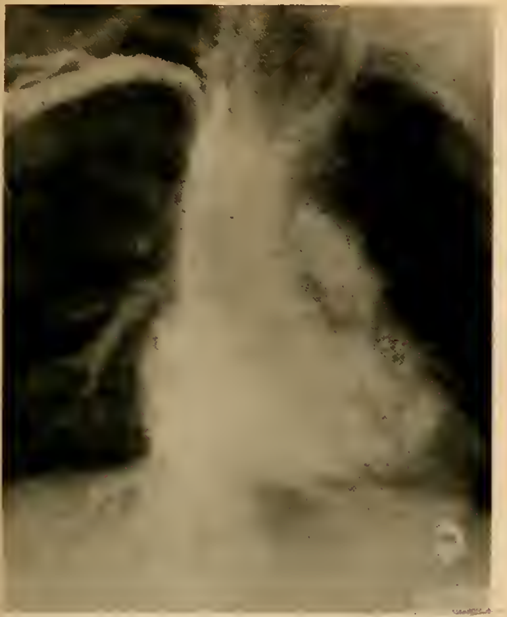

Fig. 5. Angiocardiogram (AP view of the chest) demonstrating Tetralogy of Fallot with simultaneous filling of large aorta and small pulmonary artery.

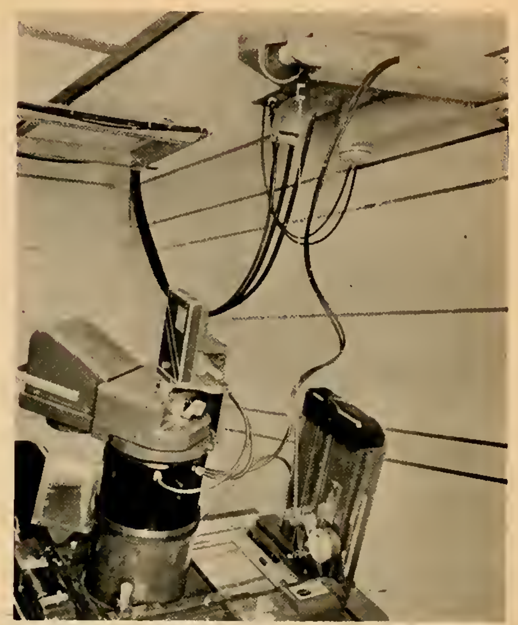

Fig. 6. Equipment used in cineangiocardiography. The movie camera (placed on top of the image amplifier fluoroscope) is synchronized with the x-ray machine to record 64 very brief exposures per second.

fants with cardiac malformations. They should be referred quickly to a cardiac center since sudden changes may occur. Diagnostic studies are usually attempted early in order to be prepared for emergency surgery. The common types are transposition, tetralogy, and pulmonary stenosis. The transpositions usually may be separated from the bulk of cyanotic conditions, since the pulmonary vascular fields appear congested in x-ray studies. Exact diagnosis depends largely on catheterization and angiocardiograms. The latter technique is particularly suited to the demonstration of right-to-left shunts.

Figure 5 shows the simultaneous filling of a large aorta and a smaller pulmonary artery as seen in the typical tetralogy of Fallot. A new refinement consists of cardiac catheterization with injection of radio-opaque material selectively into different cardiac chambers. The course of the material is filmed by cineangiography. In this fashion a small amount of contrast material is employed, multiple injections can be made, and the defects can be more exactly located. Figure 6 shows the apparatus used in these studies. It consists of an image amplifier fluoroscope, with a movie camera taking pictures of the fluoroscopic screen. In order to reduce the amount of radiation, the fluoroscope and movie camera are synchronized at 64 very brief exposures per second. The film strip can then be projected at normal speed, giving a slow motion picture of the course of the circulation.

Summary

We have attempted to outline the course to be followed by a physician when confronted with the possibility of congenital heart disease in early infancy. The management of the infant and his family depends on the nature of the murmur and the presence of cardiac symptoms and signs. Many types need immediate referral to a cardiac center for special studies because of potential dangers early in life and the possibility that surgery may be needed even during infancy.

The Spectrum of Endocardial Fibroelastosis

VICTOR M. CRESENZO, M.D.
REIDSVILLE

Recent years have witnessed increasing interest in an uncommon form of heart disease whose outstanding characteristic is extensive and marked endocardial thickening of one or both ventricles. Auricular and valvular involvement is frequently noted, and associated myocardial fibrosis is sometimes found. This extensive involvement of the ventricles, auricles, valves, and associated myocardial fibrosis is believed to be primarily responsible for the clinical picture presented. It is to be emphasized that in these cases the endocardial thickening is the primary and outstanding feature rather than the incidental autopsy finding.

Extensive involvement of the endocardium with fibroelastic changes was first described in 1816 by Kreysig (cited by St. John and Copes[1]). Since then numerous case reports have referred to this finding under a multiplicity of terms. Among these are endocardial sclerosis, congenital subendocardial myofibrosis, congenital idiopathic hypertrophy of the heart, prenatal fibroelastosis, fetal endocarditis, endocardial dysplasia, elastic tissue hyperplasia, endomyocardial fibroelastosis, elastic tissue of the myocardium, and constrictive endocarditis.

The common feature of all these conditions is the presence of obvious thickening of the endocardium. For purposes of this discussion the term "endocardial fibroelastosis" will be most frequently employed.

On reviewing the literature on this interesting entity, one is impressed with the clinical spectrum presented. At one end he finds a form seen by the pediatrician in the infant and young child which, in the past decade, has become recognized as a fairly common cardiac disease in this age group. At the other extreme is the rare type originally described by Löffler[2], in 1936, and subsequently referred to as fibroplastic endocarditis, or Löffler's endocarditis parietalis fibroplastica. In the intermediate area of the spectrum is another type occurring predominantly among adult Negroes in Africa, where it is considered one of the most common causes of heart failure. Still another type is found primarily among white adolescents and adults, mainly from North Africa and Europe.

For purposes of discussion, therefore, cases of endocardial fibroelastosis can be divided arbitrarily into several groups. Overlapping as well as distinct and specific features will be found in all groups. The groups will be discussed individually, with emphasis on the outstanding specific characteristics of each.

Group One—Infants and Children

Infants

A survey of the literature[3-15] reveals that fibroelastic endocarditis is considered by many to be one of the most common cardiac conditions associated with death in children below the age of 10 years. In his series, Diamond[3] found that one third of the children dying of congenital heart disease before the age of 10, on whom autopsies had been performed, had severe endocardial fibroelastosis. Blumberg comments further on the frequency of this fatal disease in infancy, and described 25 cases of his own observed during a five-year period.

In infants and children endocardial fibroeslastosis is seen in both the primary or idiopathic form, with or without associated congenital cardiac lesions. The most commonly associated abnormalities are congenital aortic stenosis, coarctation of the aorta, and patent ductus arteriosus. Both those cases with and those without associated congenital lesions have the common characteristic of a grossly thickened, pearly white endocardial layer. Cases of the primary type present a fairly uniform clinical and pathologic picture. Those with associated malformations are far more heterogeneous and present considerable variation in the clinical picture.

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In the average primary or idiopathic case, the clinical manifestations are sufficiently typical to alert one to a tentative antemortem diagnosis. Cough, paroxysmal dyspnea, vomiting, tachycardia, respiratory distress, tachypnea, and cyanosis occur rapidly in a newborn or young child previously regarded as normal by the parents and physician. The usually present signs of heart failure are initially attributed to bronchopneumonia. X-ray examination reveals a globular enlarged heart with bilateral pulmonary congestion. Frequently this sign may be the first indication that the heart is involved. Closer observation reveals a persistent tachycardia and muffled heart sounds. Unless some associated congenital cardiac defect is present, no significant murmurs are usually detected. The murmurs usually associated with such a lesion are not sufficiently impressive to explain the cause of the advanced failure, cardiac enlargement, and silhouette.

In addition to the tachycardia, the electrocardiogram displays a multiplicity of other nonspecific abnormalities, such as flattened or T wave inversions, prolongation of the PR interval, widening of the QRS complex, and findings of left ventricular hypertrophy and myocardial damage. Various arrhythmias with varying degrees of block, complete atrioventricular dissociation, and Adams-Stokes attacks may be evident. In his detailed analysis of 23 complete electrocardiograms from cases of primary endocardial fibroelastosis proved at autopsy, Vlad[13] noted a high incidence (16 cases) of left ventricular strain patterns in hearts with vertical or semi-vertical electrical position. Right ventricular hypertrophy was relatively common either in a pure form (4 cases) which might or might not be replaced later by left ventricular hypertrophy, or in the form of combined ventricular hypertrophy (3 cases). Alterations of the P waves were seen in 14 cases, indicating right and/or left atrial hypertrophy or dilatation.

At this stage the clinical course is unpredictable, but it may follow one of several patterns. Terminal symptoms may develop rapidly and death may occur within minutes or hours—the so-called "acute or fulminating" type of idiopathic congenital infantile endocardial fibroelastosis.

Endocardial fibroelastosis as the cause of sudden, unexpected, and unexplained death in an infant previously regarded as normal is being more widely recognized—the so-called "crib death."[12] At autopsy in such cases the unexpected finding of extensive endocardial fibroelastosis provides the only logical explanation of the cause of death, the probable mechanism being the sudden development of acute fatal arrhythmia.

On the other hand the course may be less fulminating, and there may be temporary control of periodic episodes of cardiac failure with the use of mercurials, digitalis, sodium restriction, and steroids—the so-called "chronic" type. However, the course is usually down hill, and death may result from congestive heart failure or an acute arrhythmia. Death from embolization is infrequent in this group.

In the initial phase of the disease several clinical entities may be considered in the differential diagnosis. Among the more acute bronchopneumonia and acute myocarditis of various causes. Roentgenograms of the heart reveal it to be enlarged and usually globular[1, 16]. The pulmonary markings are increased in the presence of left-sided heart failure. The cardiac silhouette is not diagnostic, but angiocardiograms provide a valuable lead toward a correct ante mortem diagnosis.

In normal patients and with conditions other than congenital fibroelastosis, angiocardiographic studies reveal reciprocal atrial and ventricular filling. Atrial systole is noted to accompany ventricular diastole, while atrial filling occurs simultaneously with ventericular emptying. In patients with endocardial fibroelastosis this reciprocity is not observed. Usually no significant changes are noted in the size of the ventricular chambers during systole and diastole, probably because of the splinting action of the thickened fibrotic endocardium. Dye injected into the ventricular cavity during angiocardiography appears to remain there. However, when similar angiocardiographic studies are performed on patients with left ventricular enlargement and failure from causes other than endocardial fibroelastosis, marked volume changes in systole and diastole are noted on the angiocardiograms. Linde[17a] cautions, however, that before the specificity of this sign can be fully evaluated, studies must be performed in other rare types of heart disease such as idiopathic cardiac hypertrophy, glycogen-storage disease, and idiopathic myocarditis.

In their angiocardiographic study of 6 patients with endocardial fibroelastosis, Lynfield and Gasul 17[b] found 3 patients with dilatation of the left ventricle and 3 with small or normal left ventricular chambers. In all of them opacification of the left side of the heart was delayed and prolonged. In 5 patients, enlargement of the left atrium was noted. Two patients showed considerable contraction of the left atrium. Those with the most severe degree of left ventricular dilatation displayed the smallest change in the size of the left ventricular chamber during ventricular systole. In one case, the chamber of the left ventricle was decreased during systole, but the residual volume was large. Therefore, it appears that in these cases of endocardial fibroelastosis with associated left ventricular hypertrophy or dilatation, chamber size during ventricular systole is either minimal or absent, with a resulting increase in residual volume.

In those patients with a normal-sized or contracted left ventricle, angiocardiograms revealed some decrease in the size of the left ventricle during systole, but again the residual volume was found to be increased. This finding of impaired distensibility of the ventricle is similar to that found in patients with certain pericardial and myocardial disease, such as constrictive pericarditis, diffuse myocardial fibrosis, nonspecific myocarditis, and amyloid infiltration of the heart[18].

In the same 6 patients studied by Lynfield and Gasul, cardiac catheterization studies of the heart revealed moderate to severe pulmonary hypertension and marked increase in the total pulmonary resistance index. Catheterization of the left side of the heart showed a markedly elevated ventricular diastolic pressure.

On the basis of their clinical, radiologic, electrocardiographic, angiocardiographic studies, and cardiac catheterization findings, Lynfield and Gasul divided their cases in two types: Type 1 is characterized by dilatation of the left ventricle and signs of left ventricular enlargement and failure, revealing evidence of marked left ventricular enlargement and hypertrophy on clinical, radiologic, and electrocardiographic examniation. In type 2, a normal-sized or contracted left ventricular cavity is found with more prominent findings of pulmonary hypertension and right ventricular hypertrophy secondary to impaired left ventricular function.

Clinical and electrocardiographic evidence of associated right ventricular hypertrophy was present, being especially prominent in the latter. In this type, the secondary effects on the pulmonary circulation are similar to those of mitral stenosis. On these same 6 patients Gasul performed catheterization studies. Catheterization of the right side of the heart showed moderate to severe pulmonary hypertension and increased total pulmonary resistance. Catheterization of the left side revealed elevation of the left ventricular diastolic pressure, indicative of left ventricular failure.

Functionally, the end result of the thickend endocardium is limitation of expansion and contraction of the ventricle. This splinting action leads to heart failure. It is postulated that endocardial fibrosis almost invariably causes hypertrophy of adjacent ventricular musculature, probably due to the increased work necessary to contract and expand the thickened fibroelastic endocardium. It is thought that the thickened endocardium interferes with and limits ventricular movements from the inside of the heart in much the same way as the scar tissue surrounding the heart in constrictive pericarditis does from the outside. Therefore the condition is sometimes referred to as constrictive endocardial sclerosis[19].

Pathologically[20], the fibroelastic changes in the endocardium vary in degree, but upon gross inspection one may observe a shiny gray-white thickening that may extend to and involve the valves. The left side of the heart is most often affected, chiefly the ventricle, where the endocardial thickening is greatest over the septal surface.

Histologically, the endocardial thickening is due to a heavy layer of elastic and collagen fibers which may extend for short distances around small blood vessels in the myocardial lesions. No specific myocardial lesions are found, although small areas of patchy fibrosis are frequently mentioned. Other changes such as calcifications underlying the thickened endocardium, and abnormal glycogen deposits are also described. The pathological changes in the lungs and other organs usually represent the effects of congestive heart failure. Mural thrombi are sometimes present and may give rise to infarcts at different sites.

Ages 2 to 6

Von Ruchian[21] arbitrarily established the ages of 2 to 16 as representing the second group in the spectrum of children and young adolescents. Endocardial fibroelastosis is much less frequent in this group than in infants and young children. The signs and symptoms may show some variation when compared with those of the infantile group. The onset is usually more insidious. The characteristic picture is that of chronic, progressive congestive heart failure, with no specific murmurs or lesions to explain the etiology. Response to digitalis, diuretics, and diet may occur but is only temporary. Complete atrioventricular block may occur, with a full Stokes-Adams syndrome. Typical anginal attacks have also been described in this age group.

Left ventricular hypertrophy is the common pattern observed. Intracardiac calcifications also have been reported. Roentgenograms, electrocardiograms, catheterization studies, and angiocardiograms are essentially like those in infants. The left atrium and ventricle are more frequently involved, with a less striking degree of endocardial thickening and cardiac hypertrophy. Mural thrombi and embolization are more common.

The course of the disease is usually protracted, with death usually occurring in congestive heart failure. Patients usually respond at first to digitalis, diuretics, low sodium diet, and other empirical measures, but eventually they become refractory to this therapy. Sudden death from complete atrioventricular block and embolization may occur.

The degree of cardiac hypertrophy and endocardial thickening is less striking than in the infantile group. It is generally believed that the etiology is the same in both age groups, the significant difference being a matter of degree, resulting in a delayed onset of clinical symptoms.

Group Two — Adults

Cases of the adult type (16 years of age and older) have sufficient clinical and pathologic dissimilarities to be divided arbitrarily, for purposes of discussion, into three groups: (1) those seen primarily in the Negro population of Africa; (2) those seen in North America and Europe, particularly in England, France, Austria, Germany, and Switzerland; (3) those with the unusual characteristics described initially by Löffler and more commonly known as Löffler's fibroplastic endocarditis.

African variety

The African variety of endocardial fibroelastosis is so called because most of the cases described have been found on the African continent. A few cases with similar features have also been reported in white patients living in these areas, and one in a North American Indian[22].

In the studies of Davies and Ball[23] performed at Uganda, endocardial fibrosis was found to be the second most common cause of cardiac failure resulting in death.

In addition to the finding of a grossly thickened, white, leathery, fibrotic endocardium lining the ventricular cavities, it was also noted that the lesion extended from the ventricular walls to the papillary muscles, chordae tendinae, and valve leaflets. Also, the diseased endocardium was frequently found to be covered with mural thrombi.

The endocardium averaged 2 to 3 mm. in thickness, but occasionally was found to be as much as 10 mm. Fibrous strands extending from the fibrous endocardial tissue and penetrating the myocardium was described in about one third of the cases. In one third of the cases an area of calcification was noted in the endocardial fibrous tissue. Superimposed subacute bacterial endocarditis was also found on the septal wall in 1 case.

Involvement of the mitral valve apparatus was noted in two thirds of the cases, varying from slight thickening to advanced involvement, where the cusps were completely embedded in a mass of fibrous tissue. This resulted in mitral insufficiency in one third of the cases and mitral stenosis in 2 cases. Bacterial endocarditis on the mitral valve was noted in 4 cases. Dilatation of the left atrium and small patches of thickened endocardium were noted in approximately 25 per cent of the cases. Right ventricular involvement was noted in 31 of the 32 hearts. Hypertrophy and dilatation of this chamber were very common. Calcification and mural thrombosis was less frequent than in the left ventricle.

Lesions of the tricuspid valvular apparatus, though minor, were noted in 26 hearts. No evidence of calcification or bacterial endocarditis were found here.

Hypertrophy and dilatation of the right atrium were present in two thirds of the cases. Focal areas of thickening were noted in one third of the cases.

The aortic and pulmonary valve was not involved in any case. The great veins entering the atrium showed no lesions.

Histopathologic studies showed most of the endocardial lesions to consist of three zones; the superficial was usually acellular and often hyalanized. Calicum deposits were frequently present in this layer. The intermediate zone consisted of loosely textured fibrous tissue. Below this was a layer of granulation tissue. All hearts showed some fibrosis of the myocardium; Davies and Ball, therefore, prefer the term "endomyocardial fibrosis."

In a comparative study of these African cases with those in his U. S. series, Thomas[20] sharply demarcated the African cases from the usual form of endocardial fibroelastosis by noting that they tend to show a destruction of the endocardium and disruption of the elastic framework by replacement fibrosis. This differs from the more uniform overgrowth of both fibrous and elastic tissue components which are so characteristic of what is usually considered to be the "typical" form of the disease. No unusual extracardiac pathology was noted other than that related to chronic congestive heart failure and terminal infection. No abnormalities were noted in the major coronary vessels, but parasitic infestation was a common finding.

It is obvious that the clinical findings in each of these African cases will be found to reflect the type of major cardiac lesions. If the lesion was predominantly one in the left ventricular endocardium, left and right heart failure appeared without any distinguishing features. In such cases the diagnosis is made only by exclusion. If the mitral valve was also involved, mitral insufficiency resulted. Davies and Ball[23] consider this the most common form of endomyocardial fibrosis and the most easily diagnosed. These patients have a high pitched apical systolic mummur and thrill in a moderately enlarged left atrium. As previously mentioned, 2 cases of mitral stenosis were also observed in this series.

North American-European

In adult cases seen in North Americans and Europeans[24-29], the usual picture is that of chronic congestive heart failure characterized by the absence of specific findings suggesting coronary artery disease, valvular disease, and the usual explainable causes of heart failure. This frequently occurs in an individual previously considered perfectly normal and healthy. The first onset of failure is frequently preceded by a severe upper respiratory infection. Once failure occurs, it usually is chronic and progressive; the relief afforded by digitalis, mecurials, and other empiric measures is only temporary. Rapid and progressive deterioration is the general rule. This entity may be compared with aortic stenosis in an adult who enjoys a normal life before decompensation develops. The subsequent course is then one of rapid, progressive, unremitting congestive heart failure. Death may also result from embolization or acute arrhythmia.

The description just given is a composite picture of the average patient in this group. Unusual cases have also been reported where the clinical picture simulated that of constrictive pericarditis and mitral stenosis only to present the typical features of endocardial fibroelastosis at thoracotomy.

Chest films usually show generalized cardiac enlargement of a nonspecific nature, with associated pulmonary congestion. Angiocardiograms again reveal a decrease of systolic and diastolic cardiac motion, with delayed opacification. The electrocardiogram shows nonspecific abnormalities consisting of conduction defects, changes of the T wave and ST segments, and various blocks. Catheterization shows elevation of pressure in the right side of the heart. The incidence of embolization is higher than in the infantile and adolescent white group.

The differential diagnosis should include the various entities which might present findings such as primary myocardial disease of viral or nonspecific origin, cardiac amyloidosis, cardiac hemosiderosis, thrombosis of the endocardial surfaces of one or both ventricles of unknown etiology, beriberi heart disease, multiple pulmonary embolization, constrictive pericarditis, and rheumatic heart disease.

The outstanding pathologic features[20] consist of a diffuse fibroelastic thickening of the myocardium and a patchy myocardial fibrosis quite comparable to that associated with the childhood type of the disease. In some cases it may be as generalized and as severe as in the childhood form; in others it may be less severe and tend toward a patchy distribution. The amount of myocardial fibrosis is about the same in both types. In some cases it may be entirely absent. Mural thrombosis, however, is a more prominent feature in the adult cases. A few lymphocytes may be noted on close observation of the myocardium, but are not sufficient to warrant consideration of an inflammatory origin.

Fibroplastic endocarditis with eosinophilia

The third type of adult endocardial fibroelastosis is that usually referred to as fibroplastic endocarditis with eosinophilia (Löffler's endocarditis parietalis fibroplastica). This condition has been reported less frequently than have the other types.

The outstanding characteristic of this group appears to be severe progressive congestive failure unassociated with any obvious type of heart disease. Valvular involvement in the presence of both systolic and diastolic murmurs is a frequent finding. The valvular lesions are usually insufficient, however, to explain the clinical picture of chronic, progressive, intractable failure.

A striking eosinophilia is one of the prominent features. In the first case reported in the American literature, by Hoffman[30] in 1956, the eosinophilia varied from 30 to 70 per cent of the peripheral white blood cell count and remained a constant finding. The bone marrow may also exhibit marked eosinophilia. The endocardial tissue is grossly thickened and fibrotic, being most prominent in the ventricles. Mural thrombi have also been described. The atria may be dilated. Cardiomegaly is present, but is not as prominent as in the other groups. Microscopic section reveals a thick mass of hyalinized connective tissue beneath the endocardium and extending for a great distance into the myocardium. No myocardial fibers are recognized within this hyalanized connective tissue.

In the differential diagnosis one considers the unusual forms of heart disease such as atrial myxoma, lymphoma, amyloidosis, nonspecific myocarditis, and hemosiderosis.

Etiology

The etiology of endocardial fibroelastosis[31 36] is obscure, and numerous theories have been suggested. In the primary or infantile type without associated congenital cardiac lesions, the many possibilities advanced include intrauterine infections or trauma, intrauterine anoxia, and abnormal diffentiation of the bulbus cordis.

It has also been suggested, but not clearly demonstrated, that endocardial fibroelastosis may be related to the collagenous group of diseases. That a genetic influence may be present is suggested by the occurrence of the disease in several siblings and twins. In his detailed study of 4 cases, Rosahn[36] describes, in addition to the endocardial lesions, a marked periarterial adventitial hyperelastosis in systemic small and medium-sized arteries, suggesting that the process is generalized and systemic, and not limited to the heart alone.

Black-Schaffer suggests that the primary defect is in the myocardium. This myocardial weakness results in a dilation of the ventricle. causing an increase in the tension on the endocardium; the proliferation of the fibrous and elastic tissue is then considered to be a compensatory response to increased pressure and stretch.

Other etiologic possibilities are metabolic. nutritional, allergic, toxic, and infectious factors.

It is apparent that no single cause can account for the multiplicity of diverse pathologic features of endocardial fibroelastosis. Thus it is reasonable to suggest that the disease represents an endocardial response to a variety of unknown or unclassified stimuli. It may therefore be considered analagous to cirrhosis of the liver, in that the microscopic studies of the end result fail to reveal the nature of the primary cause.

Summary

Endocardial fibroelastosis is one of many synonyms used to designate a form of heart disease now being more widely recognized in patients of all age groups. It is characterized by extensive thickening of the endocardium, frequently with similar involvement of the valves, chordae tendinae, and papillary muscles. On the basis of age, geographic location, and pathologic and clinical differences, one may arbitrarily divide patients with this condition into several groups. In infants it is frequently asociated with congenital cardiac lesions. It is also seen less frequently in infants without associated congenital cardiac defects. In young children and adolescents, and in adults of the North American and European continents, the type of endocardial fibroelastosis observed probably represents a lesser degree of the primary or idiopathic infantile type, with a longer survival time. The lesion here is generally characterized by a fibroelastic proliferation. The African type has been recently described as the second most common cause of congestive heart failure in that area. Its clinical picture is quite variable and its pathologic picture quite distinct from that of other types of endocardial fibroelastosis in that its main lesion is of a destructive nature.

Endocardial fibroelastosis may mimic constrictive pericarditis and mitral stenosis, and should be ruled out, if at all possible, before thoracotomy is attempted.

Numerous theories regarding the etiology of this condition are offered, but no one theory can satisfactorily explain all cases.

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Hereditary Thyroid Disease

A Report of Hyperthyroidism in Five of Six Siblings

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Although thyroid disease frequently occurs in more than one member of a family, the presence of this disease in all the siblings in one family is quite rare. I have recently had the opportunity of encountering such a family unit with 6 siblings, 5 of whom had hyperthyroidism and the sixth Hashimoto's struma. When more than one form of thyroid disease appears in a family, the question arises if a single hereditary factor can produce different disease patterns. Is the nature of the hereditary factor dominant or recessive? Since the etiology of thyroid disease is as yet unknown in the vast majority of cases, could inborn errors of metabolism and/or hereditary factors be of major etiologic importance? The cases reported here have stimulated these questions and this report. In the consideration of hereditary thyroid disease, one must naturally exclude environmental factors such as endemic iodine-lack and the high intake of goitrogenic food substances which would logically involve an entire family.

Case Reports

This family unit consists of 6 siblings whose present ages range from 34 to 49. A diagnosis of hyperthyroidism was made in 5 of the 6 between the ages of 33 and 38. The sixth had undergone a thyroidectomy at the age of 40 because of the presence of a multinodular nontoxic goiter. There were no other siblings in the family. The mother of these patients died at the age of 72 with a probable coronary thrombosis, and the father at the age of 64 with "heart trouble" and "cancer of the prostate." There is no evidence to suggest that either had had a goiter or a history of hyperthyroidism. There is a note on the chart of one of the siblings that two aunts on the mother's side had had goiters, but this could not be verified. The 6 siblings reported here have

11 children in all, aged 7 to 30, and none have any reported evidence of thyroid disease. The entire family unit was born and reared in Piedmont North Carolina and have not lived elsewhere for any length of time.

Case 1

A 33 year old white woman was found to be hyperthyroid two years after the onset of symptoms of heat intolerance, increased appetite, and excessive nervousness. When I first saw her the pulse was 110 and the blood pressure 140 systolic, 70 diastolic. The hands were warm and moist. There was no lid lag or exophthalmos. The thyroid gland was firm, slightly enlarged (estimated weight, 40 Gm.), and free of nodules. There was a 3 plus venous bruit in the right supraclavicular fossa. The heart was normal except for tachycardia and an accentuated M-1. Pulses were bounding. There was a rapid rebound in the deep tendon reflexes, and a fine tremor of the hands and tongue. The 24-hour thyroidal I^{131} uptake before treatment was 64 per cent.

The patient was prepared for operation with a five-week course of Tapazole, 60 mg. daily, and gained from 110 to 115 pounds. There was marked symptomatic improvement and significant but not complete abatement of the physical signs of hyperthyroidism. On January 7, 1950, a subtotal thyroidectomy was done by Dr. A. B. Coggeshall. The pathologic specimen weighed 37 Gm. Microscopically, "Most of the glands are well filled with colloid. In some of them the epithelium is flat; in others, it is cuboidal or columnar, and there is vacuolization of the colloid adjacent to the epithelium. Diagnosis: Diffuse epithelial hyperplasia or thyroid consistent with diffuse toxic goiter." (Dr. H. Z. Lund)

The patient's postoperative course was uneventful, without medication, and her weight has remained stable at about 120 pounds. When last seen 22 months after operation she was clinically euthyroid, with a protein-bound iodine of 4.5 micrograms per 100 ml.

Case 2

A diagnosis of hyperthyroidism was made in this patient at the age of 38 on the basis of symptoms of palpitation, increased appetite, and heat intolerance of one year's duration. The blood pressure was 120 systolic, 60 diastolic, and the pulse 120. There was no lid lag or exophthalmos. The thyroid was diffusely enlarged, grade 2 plus. There was no weakness of the quadriceps, but a fine tremor was present. Basal metabolic rates of plus 39 per cent and plus 100 per cent were obtained prior to therapy. In preparation for operation the patient was given propyl thiouracil for two months and Lugol's solution, 30 drops daily, for a few weeks. On August 11, 1952, a subtotal thyrodectomy was done by Dr. R. O. Lyday. The pathologic specimen was estimated to weigh from 50 to 60 Gm., and Dr. H. C. Lennon described the microscopic appearance as "fairly typical of involuting hyperthyroid."

I first saw the patient in July, 1959, at the age of 46, when he presented symptoms of nervousness, vague abdominal pain, and recurrent headache. These symptoms had been present since an automobile accident some eight months previously, at which time he suffered a contusion of the larynx and thrombosis of the right subclavian artery, among other injuries. The blood pressure was 150/80, the pulse 110; there was no exopthalmos or lid lag. Other findings included a well healed thyroidectomy scar, with no palpable tissue, a 3 plus venous hum in the right supraclavicular fossa, some degree of emphysema, bounding pulses except for a barely palpable pulse in the right upper extremity, and rapid rebound in all deep tendon reflexes. The 24-hour thyroidal I^{131} uptake was 70 per cent.

The patient was given a therapeutic dose of 4.0 millicuries of I^{131} orally on July 31, 1959. Four months later he showed good clinical improvement and had a protein-bound iodine level of 8.1 micrograms per 100 ml. Twelve months after treatment his blood pressure was 100/70 and the average pulse rate was 72. Vascular sounds of the neck, heart tone, and deep tendon reflexes were all normal, and there was no tremor. The protein-bound iodine was 6.1 micrograms per 100 ml., I^{131} uptake 14 per cent in 24 hours, and cholesterol was 270 micrograms per 100 ml. Clinical impression: euthyroid.

Case 3

This patient, a woman, was found to be hyperthyroid at the age of 38. Her presenting symptoms were nervousness, tachycardia, and a weight loss of 4 pounds in the face of a great increase in appetite. Physical findings included a blood pressure of 110/60, pulse 120; no exophthalmos; "fairly symmetrical enlargement of the thyroid gland, grade 2"; a fine tremor of the extremities, and definite weakness of the quadriceps. The basal metabolic rate was plus 36 per cent before treatment. She was given 300 mg. of propyl thiouracil and 30 drops of Lugol's solution daily for several weeks. On January 22, 1951, a subtotal thyroidectomy was done by Dr. Lyday, and the following microscopic description reported by Dr. Lennon: "Moderate variations in size of acini with varying degrees of colloid material within the lumen of the acini. The majority of the lining epithelial cells are cuboidal in character . . ."

There was an onset of metrorrhagia four months after operation, and the patient was started on thyroid within one year because of symptoms of hypothyroidism. She was 47 when first seen by me in May of 1960, with symptoms of lethargy, depression, weight gain, and sleepiness. Physical findings included periorbital edema, the absence of lateral margins of the eyebrows, a thyroidectomy scar, no palpable thyroid tissue, normal vascular sounds in the neck, and marked delay in the rebound of the deep tendon reflexes. The clinical impression was mild hypothyroidism. The dosage of thyroid was gradually increased to 3 grains daily, with prompt symptomatic improvement and a return of reflexes to normal.

Case 4

This man was found to be hyperthyroid at the age of 35. He had had symptoms of some months' duration consisting of heat intolerance, excessive fatigue, increased appetite, and marked nervousness. The blood pressure was 120/65, the pulse 120. There was no exophthalmos, but a slight though definite lid lag, diffuse enlargement of the thyroid ("about grade 2 plus"), and a fine tremor of the hands were noted. Basal metabolic rates of 64 and plus 69 per cent were obtained. Four plus glycosuria was present. The patient was given 300 mg. of propyl thiouracil and 30 drops of Lugol's solution per day for several months. On May 19, 1951, a subtotal thyroidectomy was done by Dr. Lyday. The pathologic specimen—estimated to weigh 40 Gm.—was described as follows: ". . . Sections of the thyroid gland reveal large areas of rather loose cellular structure in which practically no colloid material is present. Other areas show acini containing varying amounts of colloid material. Scattered about in the stroma are several typical lymphoid follicles with germinal centers."

This patient is reportedly doing very well without medication since the operation. I have not seen him personally.

Case 5

A diagnosis of hyperthyroidism was made in this patient at the age of 36, when he presented symptoms of nervousness, excessive perspiration, a 10-pound weight loss, and a definite increase in appetite. His physical findings included a blood pressure of 140/75, a pulse of 120, definite exophthalmos, a diffusely enlarged thyroid, a fine tremor of the extremities, and weakness of the quadriceps. Laboratory data were not recorded. He was given 300 mg. of propyl thiouracil daily and Lugol's solution for three months. On December 14, 1954, a subtotal thyroidectomy was done by Dr. Lyday, and the following microscopic recorded by Dr. Lennon: "Acini vary greatly in shape and size. Some are moderately well filled with colloid material, while others are rather scantily filled. There are other areas in which the acinic are quite small and contain little or no colloid material. Some of the acini show papillary infolds of the lining epithelium, which is more or less columnar in character."

This patient's siblings report that he also is doing well without medication. I have not seen him.

Case 6

This patient remembers having a goiter during her thirties. At the age of 40, on feeling "weak, rundown and moderately nervous," she saw her physician, who diagnosed a nontoxic nodular goiter. Her basal metabolic rate was then 0 per cent.

A subtotal thyroidectomy was done on April 16, 1951, by Dr. G. L. Carrington of Burlington, North Carolina. The pathologic diagnosis was Hashimoto's struma. The pathologist reviewed the sections in November, 1960, and said: "Re-evaluation of the sections of the thyroid tissue from the above patient are in accordance with the histologic pattern of Hashimoto's struma. There is the typical follicular lymphoid tissue within the gland. The glandular tissue is of the involutive type, with small acini filled with pink colloid. Acinar giant cells are present. No histologic evidence of hyperfunction of the glandular tissue, such as enlargement or hyperplasia of acinar epithelium, is present. Considerable interestital fibrosis is present and the capsule is thickened, sclerotic, and contains lymphocytic infiltration in some areas."

I saw this patient for the first time in May, 1959, when she was 48 years of age. She presented the classic signs and symptoms of hypothyroidism: lethargy, constipation, weakness, breathlessness, marked intolerance of cold, and a husky voice. Physical findings included coarse skin and hair, enlarged tongue, periorbital edema, and extreme delay in rebound of the deep tendon reflexes. She was started on small but increasing doses of thyroid, and within two months all the symptoms and signs of hypothyroidism had disappeared and her weight had fallen from 137 to 122 pounds. She was well maintained on 3 grains of thyroid per day, but on discontinuing the medication for two months early in 1960 had a return of many of her former symptoms. These disappeared again when she resumed taking 3 grains of thyroid daily.

Hereditary Aspects of Thyroid Disease

Hypothyroidism and goiter

One might expect that the clinical course of familial diseases would follow a rather uniform pattern. That this is not always the case in the spectrum of thyroid diseases is suggested by the report of Sorensen[2], who found an unusual frequency of Graves's disease, primary myxedema, and unclassified goiter in four generations of a single family. Forty per cent of the entire generation span evidenced thyroid disease, and the youngest generation, consisting of 7 siblings, included 3 with Graves's disease, 3 with primary myxedema, and 1 with a goiter, though the latter was euthyroid. Admittedly, this situation could represent the coincidental concurrence of separate familial traits. On the other hand, does it indicate that the thyroid and/or pituitary relationship is hereditarily "unstable" and that non-hereditary factors determine the eventual outcome?

More commonly, however, as in our experience, the clinical pattern is rather uniform. In the past decade there have been many references to inborn errors in thyroxine synthesis, a good number of which show a significant familial incidence. Stanbury and McGirr[3] have demonstrated that goitrous nonendemic cretinism may be explained on the basis of defective thyroxine synthesis by at least three different enzymatic defects. These authors present evidence that genetic studies support the hereditary transmission of sporadic cretinism, and the combined biochemical and genetic data suggest the influence of a single recessive autosomal gene. Pickering, Sheline, and Crane[4] report 8 children with sporadic cretinism. Sibling involvement occurred in three out of eight chances, also suggesting transmission by a recessive autosomal gene. It is postulated that in this situation hypothyroidism is secondary to inadequate, or at least ineffective, thyroid synthesis by the gland, and the appearance of a goiter at a given age depends upon a relative degree of thyroid deficiency and the ability of the gland to respond to sustained TSH stimulation. Werner, Block and Mandle[5] report abnormal circulating idoproteins in 3 members of a family, all being hypothyroid but only 2 having goiters.

Suboptimal thyroid production may be sufficiently compensated by TSH activity, however, to produce individuals who are clinically euthyroid but have goiters. Clayton, Smith and Leiser[6] describe a family unit with 4 of 6 siblings belonging to this category. These children had a demonstrable defect in organic binding of iodine. The two other siblings and the parents had no such defect and were euthyroid. These demonstrate examples of non-toxic goiters due to a familial intrinsic metabolic defect in thyroxine synthesis. Other reports of familial non-toxic goiters are available[7], but lack studies to label the actual defect in thyroxine synthesis. Finally, Beierwaltes and Robbins[8] studied a patient and his son who were euthyroid and had normal-sized thyroid glands, but had elevations of protein-bound iodine. In an analysis of the patients' thyroid status, these authors also found an elevated butanol-extractable iodine, but a subnormal red blood cell uptake of I^{131} T-3. By electrophoresis they demonstrated an increase in the thyroxine-binding capacity of the plasma thyroxine-binding alpha globulin. The patient had two other children who also had entirely normal thyroid studies.

The hereditary defect then can be gross enough to produce a non-goitrous cretin; milder, to produce a goitrous cretin; milder yet, a goitrous euthyroid; or so minimal as to affect neither the function nor the size of the gland but only isolated physiologic phenomena.

Hyperthyroidism

In 1946 Boas and Ober[9] reviewed the literature to that date and found 157 reports of hereditary thyrotoxicosis. They added the report of a family of 143 blood relatives in five generations. Of these, 11 (7.7 per cent) had proven hyperthyroidism. In addition one had a non-toxic adenoma and one had proven hypothyroidism. Neither of the latter two, however, gave a history suggestive of hyperthyroidism. Definite evidence of exophthalmos was present in all 11 thyrotoxic cases.

Cunningham and Kral[10] reported hyperthyroidism in two sets of identical twins. The first pair developed hyperthyroidism with exophthalmos at age 44. The second became hyperthyroid at age 41, but only one had typical Graves's disease. The other twin had a single nodule, was toxic, but did not have exophthalmos. After treatment with I^{131} the nodule disappeared and the patient became mildly hypothyroid. Substitution therapy was given for about two years, then stopped. Two and one half years after I^{131} therapy the patient was euthyroid, but now had mild exophthalmos. Interestingly, the second set of twins were 2 of 7 siblings, "and one other sister is also reputed of having been surgically treated for thyrotoxicosis."

The family reported by Sorenson[2] had 6 cases of proven Graves's disease and 3 probable cases. He stated the familial incidence of Graves's disease to be 20 per cent or higher. Whether hyperthyroidism is a dominant or recessive characteristic is unsettled. In the cases reported in this paper there was nearly complete sibling involvement. All 5 cases were quite typical of diffuse toxic goiter, and in none was there any question as to the diagnosis. Although the parents, aunts, and uncles were not studied, none of the 6 siblings had any recollection of a similar clinical picture in these folk. Considering the clinical severity of hyperthyroidism in the cases reported, it is quite doubtful that any of the earlier generation would have "ridden through" their illness without specific medical help. If one can assume that the same would be true of the disease process had it been present in other members of the family, the hereditary factor must be recessive; for if it were dominant, at least one, if not both, of the parents should have been hyperthyroid. Unfortunately, clinical observation and study of the parents was not available.

Hashimoto's Struma

In the cases reported in this paper, 5 of 6 siblings were hyperthyroid. The sixth had Hashimoto's struma. Was this sixth case in reality the end stage of a "burned out" hyperthyroid? Even in retrospect, however, the clinical history of hyperthyroidism could not be obtained from this patient. Spjut, Warren and Ackerman[11] studied pathologic material from 76 cases of thyroid disease that had an interval of years between thyroidectomies. Of these, 35 were Graves's disease, 22 were toxic (non-exophthalmic) goiter*, and 19 were non-toxic goiters. These authors found no evidence of progression from these entitles to or from Hashimoto's struma. Their criteria for the diagnosis of Hashimoto's struma included diffuse oxyphilic transformation of the follicular plus the lymphoid components.

In their series they did find 7 patients with a form of thyroiditis they called "severe exhaustion atrophy" (two of these had Graves's disease, 2 toxic non-exophthalmic goiter, and 3 nontoxic goiters). They draw the distinction between Hashimoto's struma and exhaustion on the basis that the latter has different histologic stages of involution and lacks the diffuse oxyphilic change in the follicular epithelium.

Sclare[12] reports 2 cases that progressed from exophthalmic hyperthyroidism to a form of chronic thyroiditis that fits this same description of exhaustion. Dunning[13] reported 3 cases in one family of Hashimoto's struma. The patients were two sisters, aged 18 and 22, and their female cousin, aged 19. The microscopic descriptions in all 3 cases fit the criteria for classic Hashimoto's struma. The recent finding of circulation antibodies to thyroglobulin in patients with Hashimoto's struma points out the need for careful clinical and microscopic evaluation of patients with "lymphadenoid thyroiditis" to see if indeed there is an etiologic difference. The hereditary role must for the moment remain obscure.

Comments

In general, thyroid disease has a definite hereditary aspect. Many patients with simple non-endemic goiter may well have siblings or other relatives with minor defects in thyroxine metabolism, insufficient to cause TSH stimulated hyperplasia. These cases would not come to light without an intensive survey of their thyroxine synthesis. Familial goitrous cretinism seems to be a well established but rather rare problem related to a specific enzymatic defect. Familial euthyroid goiters, again related to a specific enbymatic defect, have been reported from non-endemic areas. Familial hyper-

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*Some degree of nodularity was evident in specimens from all three categories. The authors separate Graves' disease from toxic (non-exophthalmic) goiter both clinically and histologically. Clinically, the duration of symptoms, age of patients, and other features suggest that "toxic (non-exophthalmic) goiters" as referred to in their report fit the clinical concept of toxic nodular goiter, but this correlation is not made clear.

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5,000	7,500 to 15,000	75.00 Weekly	114.00	57.50	152.00	76.50
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thyroidism is apparently quite frequent and the etiology is unknown. Whether or not it involves a genetically determined specific enzymatic defect must await further clarification. The problem of Hashimoto's struma is quite unclear, and the familial aspects are unknown. Perhaps, as some have suggested, the only thing inheritable is a "dysthyroidism" that may take any of the clinical forms of thyroid disease.

Summary

1. A family of 6 siblings is reported, 5 of whom had hyperthyroidism and the sixth Hashimoto's struma.
2. A review of the literature is given in reference to the hereditary aspects of goiters and hyperthyroidism.
3. A comment is made about Hashimoto's struma and the relationship between it, hyperthyroidism, and exhaustion atrophy.
4. There is a suggestion that hereditary factors may play an important, but at the moment subtle, role in a variety of thyroid disorders.

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We are sagging badly in our skill as historians. Everywhere one sees a decline in interest in hearing through a patient's whole story. The excuse is made that there is too little time available, so short-cuts and devices in history taking are sought and constructed, even though none can ever approach the performance of a knowledgeable, thoughtful, and interested physician. The art of listening and the *willingness* to listen is one of the precious assets of a fine physician. This does more to cement the physician and patient together than anything else. To listen intelligently requires depth of knowledge, imagination, an open mind, a critical sense of values, and human understanding. To do this well requires time. If we will but listen, the patient will often tell us the diagnosis, and will uncover what his illness has done to him as a person, his fears, and the tortures of his mind.—Lewis, H. P.: Reflections from our Mirror, Ann. Int. Med. 53: 1205 (Dec.) 1960.

Porphyria Cutanea Tarda

Diagnosis by Liver Biopsy

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and
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Durham

The porphyrias belong to the group of diseases referred to as inborn errors of metabolism[1], characterized by abnormal metabolism and excessive excretion of porphyrins and/or their precursors, porphobilinogen and delta aminolevulinic acid. Extensive work on all aspects of porphyrin metabolism has been done by various authors[2]. Watson[2b] classified porphyrias into porphyria erythropoetica, a hereditary light-sensitivity disorder probably inherited as a recessive trait; and porphyria hepatica, which includes (1) acute intermittent porphyria characterized by abdominal pain and/or neurologic manifestations, and excessive urine porphobilinogen and delta aminolevulinic acid; (2) porphyria cutanea tarda; and (3) a mixed type in which both cutaneous and abdominal and/or neurologic symptoms are present[3]. Recently, Watson extended his classification to include a latent and an acquired types[4].

Porphyria cutanea tarda (PCT) is a relatively rare disease characterized by easy bruising of the exposed areas of skin and formation of bullae, leaving pigmented scars; a curious livid coloration of the face and trunk, and occasionally hypertrichosis. Liver disease is seen in the majority of the cases, and almost 80 per cent of patients give a history of chronic alcoholism. Fresh liver tissue in PCT will fluoresce with ultraviolet light.

Case Report

A 50 year old white single farmer was admitted to the Medical Service of Durham Veterans Administration Hospital on March 17, 1960, for evaluation and treatment of a generalized skin rash of one week's duration. For the past year he had noticed small bullae appearing on the dorsum of his hands. These lesions would frequently discharge a clear to brownish fluid and leave brownish scars. They were sharply localized to the dorsum of the hands, not extending proximal to the wrists. He stated that slight trauma would give rise to these lesions, but no relationship to chemicals, sunlight, or anything else was noted.

Approximately one week prior to admission the patient noticed the onset of a generalized erythematous, papulo-urticarial, pruritic eruption involving the legs, palmar aspects of the hands, arms, trunk, and face. He did not recall exposure to new drugs or foods, or contact with noxious agents. He stated that his urine at times had a reddish hue, but there had been no neurologic, gastrointestinal, or other systemic symptoms. There was no family history of liver or skin diseases. He had consumed a large amount of alcohol weekly for many years. Three years ago he had a two weeks' episode of polyarthritis diagnosed by his physician as rheumatic fever, but had not had any cardiac symptoms. He had malaria in 1942 and an appendectomy in 1926.

Physical examination

Physical examination revealed a ruddy-complexioned male appearing older than the stated age of 50. There was a violaceous hue over the trunk, which blanched easily with pressure. The blood pressure was 110/70 mm. Hg. The pulse rate was 80 per minute. Periorbital edema and puffiness of the lips were noted, and the conjunctivae were injected. There was no mucosal or genital lesions. There was a diffuse giant urticarial eruption over the body, excluding the perineum and the dorsum of the hands. There were several bullous lesions and a number of brownish pigmented scars over the dorsum of both hands, not extending beyond

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From the Department of Medicine, Duke University Medical Center and Veterans Administration Hospital, Durham, North Carolina.

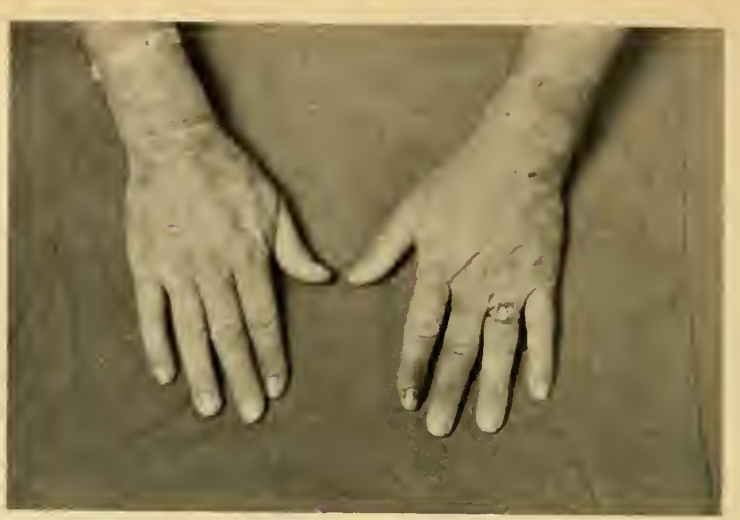

Fig. 1. A typical cutaneous bulla over the dorsum of the right hand. A number of brownish pigmented scars are also seen on both hands.

the wrists (fig. 1). Some of the bullae contained clear and others brownish fluid. The liver was enlarged to 8 cm. below the right costal margin at the midclavicular line. The rest of the physical examination was within normal limits.

Accessory clinical findings

The white blood cell and differential counts were normal. The hematocrit was 47 per cent and the hemoglobin 15.4 Gm. per 100 ml. The total eosinophil count was 90/mm³. Bleeding and clotting times were normal. Repeated urinalyses were normal. The urine was negative for porphobilinogen. Urine culture was sterile. The blood urea nitrogen was 10.5 mg. per 100 ml., fasting blood sugar 93 mg. per 100 ml., and serum chlestrol 210 mg. per 100 ml. The bromsulfonphthalein dye retention was less than 5 per cent in 45 minutes. The total serum protein was 7.3 Gm. per 100 ml., with 3.9 Gm. of albumin and 3.4 Gm. of globulin. The thymal turbidity was 4.3 units and thymal flocculation was negative. The serum alkaline phosphatase was 9.4 King-Armstrong units, and the prothrombin time was 74 per cent. Serum VDRL was nonreactive. A chest roentgenogram and electrocardiogram were normal. Repeated fresh morning collections of urine failed to fluoresce with ultraviolet light, but a 24 hour collection of urine did show fluorescence.

Two liver biopsies were performed. Fresh specimens of liver tissue from both showed bright red fluorescence under an ultraviolet light. Similar specimens obtained at liver biopsy on a number of subjects without PCT failed to show fluorescence. Liver tissue from the present case failed to manifest fluorescence with ultraviolet light after fixation, but light microscopy of the stained sections showed fatty metamorphosis (fig. 2). Histologic examination of one of the bullae of the hand demonstrated a parakeratotic keratin layer on the upper

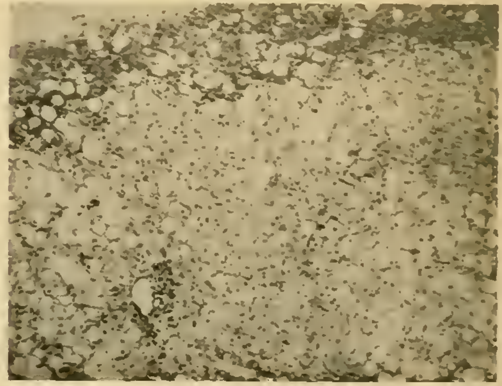

Fig. 2. Fatty metamorphosis of the liver obtained by percutaneous biopsy. The fresh tissue fluoresced with ultra violet light. (X 350)

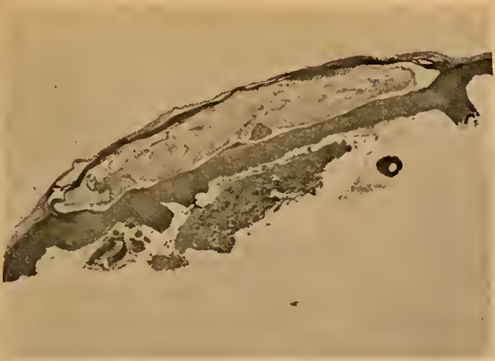

Fig. 3. Photomicrograph showing a large bulla filled with a proteinaceous amorphous material and a scattered number of neutrophils, eosinophils, and lymphocytes. The upper surface is composed of a parakeratotic layer, and underneath is an intact layer of epidermis with normal intercellular bridges and basal layer. (X 50)

surface and an intact dermis on the under surface (fig. 3). This was interpreted as a subcorneal bulla compatible with porphyria cutanea tarda.

Hospital course and follow-up

The patient's urticarial eruption did not respond to administration of Banthine and Benadryl. However, there was a rapid response to 1.5 mg. of Decadron given four times daily, with complete disappearance of the urticaria within 18 to 24 hours. Gradual withdrawal from steroids was accomplished without recurrence of urticaria. The bullae over the dorsum of the hands, however, were not affected by therapy.

The patient is being followed periodically as an outpatient. During this period he has been given a 12-week course of Dimercaprol (BAL), 300 mg. intramuscularly weekly, without any benefit to the complexion or the bullae of the hands.

Discussion

The term "porphyria cutanea tarda" was first coined by Waldenstrom in 1937[2b]. The exact biochemical derangement and enzymatic abnormalities have not been clarified. However, a possible reduction in the rate of "transformation" of uroporyhyrin and coproporphyrin to protoporphyrin has been postulated[1b]. Excretion of uroporphyrin and coproporphyrin is definitely increased, with a preponderance of the former, in this condition. Many other conditions such as heavy metal poisoning and Hodgkin's disease, also cause excessive urinary excretion of coproporphyrin, and to a lesser degree of uroporphyrin.

Recently Waldenstrom classified PCT into two types—symptomatic and hereditary. The former is thought to be secondary to hepatic malfunction such as that seen in alcoholic cirrhosis; the latter is a genetic disease. In PCT the liver has an excessive amount of uroporphyrin and coproporphyrin. Both of these two materials yield intense red fluorescence with ultraviolet light. In patients in remission, the urinary excretion of uroporphyrin is reduced to or near normal, and the excretion of copropohphyrin is also reduced but to a lesser extent. In a few of the patients' relatives, seemingly healthy and asymptomatic, similar urinary findings are reported[1a].

Porphyria cutanea tarda commonly develops during the fourth to sixth decade. Males are more often affected than females. A familial pattern of mendelian dominance can be shown in some of the cases. Recent investigations have revealed that healthy and apparently unaffected members of the family may have deranged porphyrin metabolism[1a]. Impairment of the liver is commonly seen. Many authors have stressed the importance of hepatotoxic agents and specifically alcohol [1a, 4]. Tio and others[5] reported a case of PCT in an 80 year old woman in whom the onset of cutaneous bullae had occurred during the preceding year. She was found to have a hepatoma which fluoresced and contained considerable amounts of porphyrin. Following operation the serum, which previously had fluoresced under ultraviolet light, failed to fluoresce.

Skin lesions are usually the presenting problem. They are usually characterized by the formation of low grade blisters and a tendency for the light-exposed areas to erode with melanosis, hypertrichosis, and violaceous coloration. Hyperpigmentation, diffuse or mottled, may be present. Sclerodermoid yellowing and hardening of the exposed skin is not uncommon.

Histopathologic changes of the cutaneous lesions are not distinctive, and can be identical to those of epidermolysis bullosum. Differentiation of these two conditions may be extremely troublesome[6]. Bruntsing[2a] studied 10 cases of PCT and gave a detailed description of the lesion. The bullae are of the tension type, without acanthosis, and are usually subepidermal. The overlying layer is thick and shows parakeratotic changes. Microscopically, melanosis and deposition of an amorphous material can be found. The remaining picture is similar to the skin chronically exposed to light. Demonstration of increased urine porphyrins and fluorescence of biopsied liver tissue would easily differentiate PCT from epidermolysis bullosum.

There is no specific treatment for this affection. Patients should be advised to abstain from overindulgence in alcoholic beverages and to protect the skin from heat, physical trauma, and sunlight. Improvement with dimercaprol (BAL) and sodium ethylenediamine tetra acetic acid (EDTA) has been reported[7], but not confirmed. Our patient was not affected.

Summary

A case of porphyria cutanea tarda in a 50 year old chronic alcoholic was diagnosed on the basis of clinical findings and proved by fluorescence of liver biopsies and a 24 hour collection of urine. The patient failed to show any improvement after receiving 300 mg. of BAL weekly for 12 weeks.

Acknowledgement

We gratefully express our thanks to Mr. Leonard Hart, Medical Illustrations, Durham VA Hospital, for the photography in this manuscript; to Ernest Chick, M.D., Pathologist, Durham VA Hospital, for evaluation of the liver section, and to E. Harvey Estes, M.D., Chief of Medicine, Durham VA Hospital, for his encouragement and advice.

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A large, perhaps growing, percentage of the population is not making plans for the future through savings or private insurance, in part because during the depression a philosophy was widely promoted that it is an economic crime to save. People should spend as fast as possible everything that they can earn, borrow or otherwise acquire. The Government will somehow provide needed medical care when illnesses come along.—Judd, W. H.: Physicians in a Changing World, New England J. Med. 263: 895 (Nov. 3) 1960.

Dimethpyrindene In Pruritus — A Clinical Appraisal

S. F. HORNE, M.D.

ROCKY MOUNT

One of the most common symptoms encountered in the practice of dermatology and the one most frequently requiring treatment is pruritus. Whether associated with an inflammatory lesion, an allergic phenomenon, or the skin manifestations of a systemic disease, pruritus must be controlled for the patients' comfort while slower, more definitive therapy is being instituted.

For many years it has been necessary to employ a variety of lotions, ointments, creams, compresses, and baths. These topical measures were neither aesthetic nor highly effective; the difficulties of diffuse, frequent application left much to be desired, and the antipruritic preparations occasionally caused a contact eczematous dermatitis.

A significant achievement was made with the development of certain phenothiazine derivatives that proved orally effective in the symptomatic control of pruritus. Trimeprazine and methdilazine are examples of this group of compounds. Continued clinical experience[1] with these agents has shown that, while effective in relieving the pruritus, they may produce drowsiness, vertigo, nausea, vomiting, and neuromuscular reactions resembling the extrapyramidal symptoms seen in parkinsonism. More disturbing, particularly when one considers that this treatment is only symptomatic, are instances of toxic effects on the hematopoietic system[2] (agranulocytosis) and liver.

The development of a potent, antipruritic new compound, dimethpyrindene,* that is not related to the phenothiazines is timely. In numerous trials, dimethpyrindene has been shown to be free of any toxic effects on the liver or hematopoietic tissues[3]. These encouraging results suggested that a study of the effectiveness of dimethpyrindene in controlling pruritus was in order.

Materials and Method

One hundred and five patients of both sexes (table 1) with a variety of dermatologic lesions giving rise to pruitus were studied. The group consisted of 45 males and 60 females, with ages varying from 3 months to 86 years.

Table 1

Sex Distribution of 105 Patients With Pruritus

Diagnosis	Male	Female
Psoriasis	1	4
Neurodermatitis	20	30
Lichen planus	2	3
Solar dermatitis	3	—
Atopic dermatitis	7	9
Tinea cruris	4	3
Pityriasis rosea	4	9
Dermatitis herpetiformis	1	—
Dermatitis venenata	3	2
TOTALS	45	60

In the adult patients, the initial dosage of dimethpyrindene was 1 mg. and in infants 0.3 mg. four times daily. This dosage was increased as necessary to control the symptom.

Because of the difficulty in evaluating degrees of pruritus and results of treatment objectively, the medication was considered effective only when the patient was not troubled with pruritus between doses.

Results

The effect of the drug became evident 30 minutes to one hour after ingestion and persisted for four to eight hours. The final dosage employed in the patients noted in table 2 varied from 1.2 mg. to 15 mg. per 24 hours.

With the initial dosage it was possible to obtain satisfactory relief of pruritus in 65 (64 per cent) of the patients. By readjusting the dosage individually, good control was obtained in another 14 patients (14 per cent). Dimethpyrindene relieved the symptom of pruritus in 79 of 105 patients (78 per cent).

It is important to note that these results

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*Dimethpyrindene supplied as Forhistal by the Medical Research Division, CIBA Pharmaceutical Products, Summit, New Jersery.

Table 2

Results of Treatment with Dimethpyrindene

DIAGNOSIS	Total No. Patients Treated	No. Effectively Controlled	No. Unaffected or Incompletely Controlled
Psoriasis	5	5	—
Neurodermatitis	50	35	15
Lichen planus	5	4	1
Solar dermatitis	3	2	1
Atopic dermatitis	16	12	4
Tinea cruris	7	5	2
Pityriasis rosea	13	13	—
Dermatitis herpetiformis	1	—	1
Dermatitis venenata	5	3	2
Totals	105	79	26

Treatment effective in 79 of 105 patients (78 per cent)

apply only as regards pruritus. Thus, while the pruritus associated with lichen planus was controlled, the basic lesion was not affected. The relief to the patient was dramatic, however, and afforded comfort while slower definitive therapy was instituted.

In 19 patients it was possible to compare the results of dimethpyrindene to trimeprazine. In 2 patients of this group, neither agent was effective in relieving pruritus. Six of the patients preferred dimethpyrindene to trimeprazine, 6 expressed the reverse opinion, and 5 had no preference between these compounds.

Side effects

In the group of 106 patients, only one side effect was noted. This occurred in a 68-year-old white woman, who experienced headaches while taking the drug. The headaches disappeared within 24 hours after the medication was discontinued. No other side effects or toxic reactions were observed.

Discussion

While it is widely acknowledged that patients must be afforded relief from annoying pruritus, it must be borne in mind that symptomatic therapy ideally should be: (1) effective; (2) convenient and aesthetic; (3) free of undesirable side effects; (4) free of toxic effects.

Those phenothiazine derivatives with antipruritic action, while satisfying the criteria with regard to effectiveness and convenience, are not satisfactory in terms of side and toxic effects.

Dimethpyrindene, a new indene compound possessing potent antihistaminic activity, has proved at least as effective as the phenothiazine derivative trimeprazine, and has shown itself to be remarkably free of side effects, particularly the more serious ones occasionally encountered with phenothiazine agents. These factors suggest that dimethpyrindene more closely approximates the criteria of an ideal agent for the symptomatic control of pruritus.

Summary

1. Dimethpyrindene was orally effective in controlling pruritus in 79 of 105 patients with varying dermatologic disorders giving rise to this disturbing symptom.
2. Dimethpyrindene was remarkably free of side and toxic effects, even in dosages as high as 15 mg. per day.

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A Tribute To Dr. A. C. Bulla

J. W. R. Norton, M.D., M.P.H.*
Raleigh

The dedication of the A. C. Bulla Health Center brings to focus the work of a dedicated person and the progress of public health in this community.

It is a matter of gratification to all of us who know Dr. Bulla that Wake County saw fit to name this health center in honor of the man who for more than 37 years was Wake County Health Director. Into this beautiful and excellently equipped service institution went the long years of labor and the farsighted dreams of this leader in public health.

Wake County is aware of the quality of its leaders in many fields. Such men as former chairman John Swain and Ben Haigh, the present chairman of the Board of County Commissioners, are examples of this leadership. They did well to plan with the Wake County Board of Health for such an adequate health center and for the recognition being given by naming it for Dr. Bulla.

Dr. Bulla was a leader who was wise in his choice of a health staff. Such people as Flora Wakefield, Dewey Sanderford, Anna Johnson, and the present health director, Dr. Isa C. Grant, are examples of the continued high quality of staff personnel.

Dr. A. C. Bulla, the son of a physician, was born in Randolph County about seven miles from Asheboro. Two of his uncles were also physicians. One of these is still practicing in Randolph County, though now 98 years of age.

Dr. Bulla received his degree in medicine from the Medical College of Virginia in 1915. He then began work with Dr. George Cooper, Sr., of the State Board of Health, and for two years gave immunization all over North Carolina. Then, beginning in 1917, Dr. Bulla served for four years as health director in Forsyth County. In November, 1921, he began the long and fruitful service as Health Director of Wake County, and continued in this position until his retirement in July, 1958.

Milestones in Public Health

Look with me at some of the health programs initiated in Wake County by Dr. Bulla.

He emphasized health services for school-age children and began the dental health program for school and pre-school children. He was a pioneer in the successful movement toward fluoridation for Raleigh.

He instituted the dairy inspection program in the county, and the environmental sanitation program which has been beneficial in the elimination of many intestinal diseases.

In the area of maternal-child health, he sought to give adequate training to midwives and to increase concern for prenatal care and added facilities and help for the well child.

He organized a Department of Vital Statistics which has been able to give complete services to the citizens of Wake County so that they may have a sound base for the evaluation of their health programs.

Dr. Bulla recognized the economic waste and hazards to human beings which come about when there are inadequate rabies and rodent control programs.

The health service programs that were in operation when Dr. Bulla came to Wake County he continued to implement and to strengthen as needed. He recognized the demands put upon the health department by the growth of the population and by the discovery of the effectiveness of new programs. To meet these needs the staff grew from 5 people to a total of 39 at the time of his retirement. The budget in 1921 was $20,000. The budget at the time of his retirement was $201,000, a little more than 10 times his first budget.

Progress that was little short of amazing came in the period of Dr. Bulla's service. Great strides were made in tuberculosis control, the virtual elimination of typhoid,

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Read at the dedication of the A. C. Bulla Health Center, Raleigh, North Carolina, March 5, 1961.

*State Health Director, North Carolina State Board of Health.

smallpox, malaria, diphtheria, measles, and other plagues formerly prevalent among children.

Dr. Bulla realized that prevention is the best investment. He stepped up every program that approached the health of the community from a preventive standpoint. He stressed testing programs in every area where they would lead to the discovery of disease, then referred these cases to private physicians to treat. These programs included cancer control, virus immunizations, the testing and preventive approach to diabetes, accident control, and mental health.

These are only a few illustrations out of the long and helpful service Dr. Bulla has rendered not only to Wake County but, by example and inspiration, to public health far beyond this county's borders.

Diplomacy and Education

One citizen of Raleigh, who was on the Wake County Board of Health from its beginning until 1941, said:

"You can't put on paper the things that Dr. Bulla has done. Statistics won't tell the true story. You would have had to work with him to understand it. The key words in Dr. Bulla's success have been *diplomacy* and *education*. He never tried to drive things through. He always dealt with them in a reasonable way and never attempted to use undue pressure. He educated and carried the public along with him in his program. He educated the County Commissioners and the City Commissioners in his program, but it was all done slowly and quietly."

No such accomplishments could be brought about except by a person dedicated to his task, a person with outstanding qualities of hand and mind and heart.

Dr. Bulla was a man who faced work confidently and with a smile. Perseverance could have been his middle name.

I know something of his skill, for I have received my typhoid shots from Dr. Bulla. Contrary to some experiences, he was able to administer the shots almost painlessly. My sister Anne claims that he promised her a gold-tail monkey if she did not cry when she received her typhoid shot, and she still considers that she has claim on Dr. Bulla.

Dr. Bulla added to his fine training and experience by wide but selective reading, by attending and participating in meetings of importance in his work, and by discussion with persons in widely divergent disciplines and interests.

If one were to choose five words, each of which would delineate some aspect of Dr. Bulla's character and work, these words would come to mind—*Dedication, Unselfishness, Gentleness, Patience, Teamwork.*

Fortunately, as we are assembled here to dedicate the A. C. Bulla Health Center, Dr. Bulla himself can be with us. Still active, a wise elder statesman, he continues to contribute, and the service of his health center and its well qualified staff will see Dr. Bulla's spirit living on his co-workers.

Rarely is it possible for an occasion such as this to combine so many memorable and worth-while facets of life as the tribute we pay and the future we view in naming this facility, the A. C. Bulla Health Center.

Report from The Duke University Poison Control Center

Jay M. Arena, M.D.
Director

PHOSPHATE ESTER

A group of highly toxic phosphate esters discovered by the Germans during the second World War in their search for lethal gases have come into widespread use throughout the world for eradication of certain insects, particularly the soft-bodied ones. With the exception of malathion and chlorothion, these insecticides have high mammalian toxicity, and are readily absorbed from the skin, lungs and gastrointestinal tract, thus increasing the possibility of acute poisoning during their application and use.

All the organic phosphate insecticides have essentially the same mechanism of action. They owe their toxicity to an ability to inhibit cholinesterase, the enzyme that normally detoxifies acetylcholine. Some organic phosphates undergo metabolic conversion, to yield cholinergic metabolites. Thus the insecticidal thiophosphates undergo metabolic change in the liver consisting of replacement of sulfur by oxygen. The resulting oxygen analogue is the inhibitor of cholinesterase. The end result of the inhibition of cholinesterase activity is accumulaation of acetylcholine. The resulting symptoms resemble those that would be expected to result from excessive and continued stimulation of the central nervous system and structures innervated by the parasympathetic and somatic motor nerves.

The clinical manifestations of poisoning consist of weakness, unsteadiness, blurred vision, and a sense of constricture of the chest. These symptoms are followed by vomiting, abdominal cramps, diarrhea, salivation, lacrimation, profuse sweating, tremor of the extremities, and difficulty in breathing. Pinpoint and nonreactive pupils are observed, and secretions accumulate in the respiratory passages. Cyanosis as well as severe muscular fibrillations, coma, and convulsions may ocur. Death is associated with respiratory failure. After lethal doses the onset of symptoms usually is extremely rapid, and death generally occurs within 24 hours. After sublethal doses, the inhibition of cholinesterase is reversed and the symptoms disappear, but the rate varies from a few hours to several days with different compounds.

The medical aspects of poisoning by insecticidal organic phosphates consist of prevention, detection and treatment. Prevention can be accomplished by educating people occupationally exposed to the compounds (farmers and florists) concerning the dangers of contact, inhalation, and ingestion. Protective clothing should be worn, and contaminated skin washed immediately.

Detection of organic phosphate poisoning depends on recognition of the characteristic symptoms of acetylcholine poisoning. Exposure to amounts too low to produce symptoms can be detected by periodic measurement of blood cholinesterase. Marked depression of the cholinesterase activity of the blood occurs before symptoms appear. Since a drop in enzymatic activity to 30 per cent or less of normal is associated with signs of poisoning, it is thus possible to terminate the exposure or institute proper protective measures before poisoning occurs.

The present method of treating acute cases consists of supporting respiration and administering atropine. Artificial respiration or the administration of oxygen, when possible, should be maintained as long as signs of respiratory embarrasment persist, and the respiratory tract should be kept clear of secretions. Atropine is the specific antidote for this poison. The initial dose usually is 2 mg., but in severe cases 4 or 6 mg. can be given intramuscularly. Initially the interval between doses is 15 to 30 minutes; repeated doses are given less frequently. A total dosage of 20 to 30 mg. usually is required during the first 24 hours. Although atropine acts specifically against the parasympathetic effects of the poison, it does not help the muscular weakness. Therefore, artificial respiration may be required for many hours. The airways should be cleared by suction and positive pressure oxygen given if pulmonary edema occurs. The use of a respirator may prove lifesaving. Respiratory depression contraindicates the use of morphine. Pronounced changes in electrolytes and serum pH may require the administration of adequate amounts of calcium and sodium bicarbonate intravenously.

The intravenous administration of 1,000 to 2,000 mg. of pyridine-2-aldoxime (2-PAM) or diacetyl monoxime (DAM) will alleviate the generalized weakness produced by quaternary ammonium or organo-phosphorus anticholinesterase compounds in normal subjects and in patients with myasthenia gravis. These oximes are valuable adjuncts to atropine in the management of anticholinesterase intoxication and should diminish the necessity for, or duration of, artificial respiration and endotracheal intubation in phosphate ester poisoning. Protopam chloride (2-PAM Chloride) can be obtained from Campbell Pharmaceuticals, Inc., 254 West 31st Street, New York 1, New York.

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JULY, 1961

SPECIAL WEEK ON AGING

Someone has said that there are now more than 52 "special weeks" every year. Most of these are frankly commercial—for example, "Drink More Milk Week," "Eat More Apples Week," and the like.

One special week, however, is not only free from any taint of commercialism but is as nearly altruistic as one could possibly be. This is the week of July 16-22, which Governor Terry Sanford has designated "as a period for particular emphasis on and attention to the interests, problems, and desires of our older citizens."

The full text of the Governor's statement is published elsewhere in this issue. It should have a special appeal to medical men, since they are vitally concerned with the well-being of our older citizens.

KEFAUVER'S DRUG BILL

Dr. Morris Fishbein's long experience as a member of the Council on Pharmacy and Chemistry of the American Medical Association and the United States Pharmacopoeial Commission certainly qualifies him as an expert witness in the case of Kefauver versus the Pharmaceutical Industry. In an editorial from the *Medical World News* for May he discusses the Kefauver-Celler bill to regulate the licensing, patenting, promotion, and naming of new drugs. Although some would not agree with him that the use of generic names for new drugs instead of the multiplication of brand names would do as much harm as good, nearly all doctors, would agree whole-heartedly with everything else he said in the last half of the editorial, which is quoted below:

> The height of folly is the proposal in the Kefauver doctrine that manufacturers be required to provide physicians "with clearer, better, and additional information on the bad as well as the good effects of drugs." The Secretary of the Department of Health, Education and Welfare would be required to mail regularly to all physicians brochures containing lists of drugs potentially dangerous or harmful. It is also suggested that "he may include in that list such information relating to those dangers or harmful effects as the Secretary may consider in the best interest of public health."
>
> Finally, the legislation says, "the Secretary shall have authority to determine the name of any drug as he shall find necessary or desirable in the interest of usefulness and simplicity." This shall thereafter be the official name of that drug. Furthermore, "no official name shall be given to any drug product which is a combination of two or more drugs." One might as well give the Secretary of HEW the responsibility for naming all the babies born in the United States.
>
> Whoever conceived this utterly preposterous proposal did so without the slightest experience or knowledge in the field concerned. Regardless of its constitutionality, the maleficent effect on any industry needs no emphasis.
>
> Unfortunately this legislation was assigned after its introduction to Sen. Kefauver's committee for hearings. Thus, the investigator who acted as prosecutor now becomes his own judge.

As regards patent legislation, Kefauver purposes that a three-year limit be set on drug patents, while disregarding the 17-year period in other fields. The Senator's ideas are likely to inhibit any company from creating new drugs, since it will make it impossible to recoup the initial investment during the brief period in which the holder of a patent will be entitled to have exclusive use. Furthermore, the proposed legislation would deter any company from attempting to bring out its own formula or modification of an existing drug. The effect would be to stultify new drug development.

* * *

A STRONGER STAFF OF LIFE

On February 27, 1961, the American Bakers Association celebrated the twentieth anniversary of bread enrichment with a meeting in New York City. The principal feature of the meeting was an address by Dr. W. H. Sebrell, Jr., Director of the Institute of Nutrition Sciences, Columbia University. Copies of this address may be obtained by writing to the American Bakers Association, 20 North Wacker Drive, Chicago 6, Illinois.

Dr. Sebrell told, in fascinating style, the story of the various means used to enrich our daily bread—beginning with the synthesis of vitamin B by Dr. Robert Williams after 26 years of research. Before the so-called enrichment program went into effect, vitamin deficiency diseases, pellagra, beriberi, and ariboflavinosis were widespread—also mild anemia due to insufficient iron.

As a result of cooperation between the National Research Council, the United States Department of Agriculture, the United States Public Health Service, and the American Bakers Association, a formula was evolved for enriching bread with the necessary vitamins and iron. In May, 1941, the United States Food and Drug Administration set legal standards for minimal and maximal amounts for the nutrients which could be included. The American Bakers Association spends four million dollars a year or more on enrichment—but the sale of 12 or 14 billion pounds of bread brings the cost to a fraction of a cent a loaf. Dr. Sebrell points out that enrichment does not mean adding to the calories of bread, but to the vitamin and iron content.

The program of enrichment has resulted in a dramatic improvement in the health of the nation. Pellagra, beriberi, and other deficiency diseases are now so rare that many younger doctors would have difficulty in recognizing them.

As Dr. Sebrell concludes his address: "It is a wonderful thing to know that in our daily bread today we have a product which has a vital role in our nation and its health—a food product so universal in its acceptance and usage that at any time it can be called upon quickly, easily and inexpensively to improve the nutrition of the masses of people either by correcting a dietary deficiency or to improve health."

The American Bakers Association deserves great credit for giving the American people a much stronger staff of life to lean on.

* * *

ACCIDENT NEUROSIS

Some eminent neurologist said that he had never known a traumatic neurosis to result from an injury sustained by an individual working for himself. Certainly one of the saddest manifestations of the frailty of human nature is the long time so many people require to recover from accidents when they are being paid for time lost.

In the Milroy Lectures for 1961, published in the *British Medical Journal* for April 1 and April 8, a British neurologist, Dr. Henry Miller, gives at length his own experience based upon 4,000 patients "examined for medicolegal assessment after accidents during 12 years of consultant practice." Dr. Miller found that accident neuroses were more apt to occur after relatively slight than after severe injury. His statistics showed that unskilled or semi-skilled workers were far more apt to develop accident neuroses than those of higher status. He noted that the subjective symptoms were grossly exaggerated during the period of compensation, though objective signs of anxiety were relatively uncommon. Patients refused to admit any improvement in their symptoms as long as the claim for compensation was not settled. Finally, in a follow-up study of 52 cases two years after settlement, Dr. Miller found that only two were still disabled by their psychiatric symptoms. "Both instances were characterized by diagnostic confusion, substantial lump-sum payments, and continuing National Insurance pensions for the results of the accident. In three other cases psychiatric symptoms persisted without occupational disablement: in each instance similar symptoms had been present for many years before the accident."

Any veteran physician could have predicted the lesson to be drawn from Dr. Miller's lectures: the sooner the case is settled, the better for the patient, the doctor, the family, and everyone except the lawyer.

* * *

WHAT PRICE AIRCRAFT SPEED?

Although statistics show that airplanes offer much safer transportation than do automobiles, the increasing number of aircraft accidents is a cause for real concern. A series of recent air crashes in New England prompted the editor of the *Rhode Island Medical Journal* to comment on it in his leading editorial for April. The editorial reminds us that last year 700 people had been killed in passenger plane accidents. Its final paragraph is so pertinent that it is quoted in full.

"Even though air travel is undoubtedly one of the safest activities in which man engages, the apparent likelihood of total destruction when failure occurs gives such failure a peculiarly chilling aspect. As layment in these extremely complex and technical matters, we cannot hope to offer substantially constructive suggestions. Yet as doctors we cannot stand aside in the face of such appalling loss of life. We suggest, perhaps timidly, that a new approach to the problem of air safety be sought. Without taking a horse and buggy posture, it may be seriously questioned whether further increases in the speed of commercial aircraft, with the resulting geometric multiplication of problems, can really be justified; and if it can, whether a limit must not eventually be established. May there not be a limit to the number of flights allowable in any given time and area? We further wonder whether, if electronic traffic control, three-dimensional radar, and enlarged and better airports are necessary, they should not be procured on a crash basis without regard to expense. Although budgetary matters cannot be ignored, we feel they have little substantial validity where human lives may be unnecessarily sacrificed. If techniques and equipment are still under investigation, money for the necessary research should be spent without hesitation. If new seating, seat belts, and other accessories are shown to be advisable, modifications should not await the obsolescence of present equipment. We feel that many of these measures are legitimate areas for federal spending when it may spare the air lines at times prohibitive costs. These are some of our thoughts on a problem about which we must not assume a fatalistic outlook."

* * *

MISPLACED SYMPATHY

It is hard to understand the length to which some people will go to attract attention and especially to win sympathy. A few weeks ago a woman gained wide publicity by saying that she had walked 27 miles from her home in Surry County to visit her 9 year old son who was a patient in the North Carolina Baptist Hospital in Winston-Salem. The story made headlines in the daily papers of New York and other large cities, and was even picked up by the London press. The truth was that she walked less than a mile to the home of an accommodating neighbor, who drove her to Winston-Salem, where she told a chaplain that she had walked the entire distance.

Unfortunately, her widely disseminated lie will never be overtaken by the truth, and our Yankee friends will have one more story to spread about the backwardness and general cussedness of the South. Truth may often be stranger than fiction—but it is not nearly so swift a traveler.

President's Message

It is going to be our effort this year to give the State Medical Society a good sound administration with just as little "confusion" as possible with reference to many of the legislative matters which are coming up both in the State Houses in Raleigh and Washington. We propose to keep you informed as to what is going on and what legislation is being brought up. We must impress upon the public that the Kerr-Mills Law can do the job that needs to be done and do it quickly, effectively and economically.

The Kerr-Mills Law deserves a chance to prove its worth. It must not be thrust aside without fair trial in favor of unsound and dangerous alternatives. Each doctor who reads this is urgently requested to write his United States Senator and Congressional Representative in Washington, D. C. and ask that they oppose any effort to saddle the nation's elderly with a rigid program of compulsory national health insurance.

The health care of the nation's elderly is at stake, and regardless of the age of your patient, his or her health care is equally at stake, for socialized medicine for the aged will one day mean socialized medicine for every American—including your patient.

If you value your physician's freedom to safeguard your health without government interference, urge your Representatives in Washington to give the Kerr-Mills Law a chance and to oppose Forand-type legislation.

Below we are giving to you a scale with reference to life expectancy. This was taken from the Annals of the American Academy of Political and Social Science—National Office of Vital Statistics—National Resources Committee. The figures are accurate.

Expectation of Life in Years
White race

	Ages			
	0	**20**	**45**	**65**
1900-02				
Male	48.2	42.2	24.2	11.5
Female	51.1	43.8	25.5	12.2
1929-31				
Male	59.1	46.0	25.3	11.8
Female	62.7	48.5	27.4	12.8
1939-41				
Male	62.8	47.8	25.9	12.1
Female	67.3	51.4	28.9	13.6
1949-51				
Male	66.3	49.5	26.9	12.8
Female	72.0	54.6	31.1	15.0
1955				
Male	67.3	50.1	27.3	12.9
Female	73.6	55.8	32.1	15.5
		Non-white		
1955				
Male	61.2	45.5	24.8	13.2
Female	65.9	49.6	27.9	15.5

This spectacular rise in life expectancy in the present century was shared by both men and women.

To whom should the credit for this increase in life expectancy go? Certainly the federal government has done nothing to help it. Instead the credit should go to the medical profession of the United States with its allied interests.

The drug firms certainly deserve lots of credit for the vast amount of research work they have done in producing the so-called "wonder drugs". The commercial drug firms have spent an untold amount of money in experimental and research work and in recent months have been seriously harassed by a congressional committee for their fine work.

We so often hear and read so much about the pitiable plight of the poor old man of 65. However, medicine and its allies, as pointed out by Dr. Askey, President of A.M.A., have a health team which will continue to improve the health status of our aging population. More people will live more years over 65 free of any serious and disabling illness. They will enjoy these added years because of what medicine can and will do for them.

Many of our men, 60 and 65, are retired entirely too early. They are retiring when they are reaching the peak of their efficiency and still able to carry on for many years. When they retire they are put out of circulation and their trend is always downward. They lose their desire to do good and use their talents.

Claude B. Squires, M.D.

Committees & Organizations

Governor's Coordinating Committee on Aging

GOVERNOR SANFORD'S STATEMENT ON SPECIAL WEEK ON AGING

Special consideration will be given to the health, happiness and welfare of North Carolina's older population during the Special Week on Aging, July 16-22, 1961.

Our State is proud of its more than 300,000 citizens 65 years of age and older, and it is with great pleasure that we honor them during this, their special week of recognition.

Each year brings a sizeable increase in the number and proportion of this segment of our population. Many factors enter into this change.

With this increase — and I must say welcome increase — in our older population segment, come the great responsibility and moral obligation to heed the needs of this group. Our attention must turn not only to their physical and economic needs, but also to their various other needs.

In cooperating with the Governor's Coordinating Committee on Aging, North Carolinians should work toward a better understanding of the problems of our aging. We must join forces at local, state and national levels to meet the challenge of this age group and to work thoughtfully toward the most beneficial results.

It is my pleasure, therefore, to designate the week of July 16-22, 1961, as a period for particular emphasis on and attention to the interests, problems, and desires of our older citizens.

I urge all my fellow citizens to observe this week in every appropriate way. I hope that individuals and organizations will show sincere concern in expanding the opportunities for the aging to participate in community affairs and to become better acquainted with the existing facilities which are available to them.

Let each of us express our personal appreciation for the many contribution's of our older citizens to our way of living and to the important resources of North Carolina.

Bulletin Board

COMING MEETINGS

Duke Medical Postgraduate Course—Morehead-Biltmore Hotel, Morehead City, July 17-22.

Southern Obstetric and Gynecologic Seminar—Grove Park Inn, Asheville, July 17-22.

Tri-State Medical Association of Virginia and the Carolinas—Virginia Beach, July 27-28.

Institute on the Problems of Tuberculosis—Lake Junaluska, August 20-23.

Forsyth County Twelfth Annual Heart Symposium—Hotel Robert E. Lee, Winston-Salem, September 22.

Eleventh Annual Postgraduate Obstetric Pediatric Seminar—St. Petersburg, Florida, August 17-19.

Blue Ridge Chapter of General Practice, Virginia Academy of General Practice, Annual Summer Symposium: Inflammation; Mental Depression—Hotel Roanoke, Roanoke, Virginia, July 22.

American College of Gastroenterology, Annual Postgraduate Course—Sheraton-Cleveland Hotel, Cleveland, Ohio, October 26-28.

Interstate Postgraduate Medical Association of North America, 1961 General Assembly—Madison, Wisconsin, November 13-16.

News Notes from the Duke University Medical Center

Dr. Barnes Woodhall, dean of the Duke University School of Medicine, has been appointed chairman of the Veterans Administration Special Medical Advisory Group.

This committee consists of medical educators and scientists. It was established by Congress to advise the Veterans Administration on policy and professional matters related to the medical treatment of veterans.

Dr. Woodhall has served as vice-chairman of the group during the past year and has been a member since 1958.

* * *

Richard M. Conover, supervisor of inhalation therapy at the Duke University Medical Center, has been elected president of the newly formed North Carolina—South Carolina Chapter of the American Association of Inhalation Therapists.

Serving with Conover during the coming year will be Roy E. Taylor, supervisor of inhalation therapy at Anderson, South Carolina, Memorial Hospital, vice president; and Mrs. Gwendolyn S. Salmon, senior technician in the Duke cardiopulmonary laboratory, secretary-treasurer.

The Board of Advisors for the two-state chapter is composed of Dr. C. Ronald Stephen, professor and head of the anesthesiology division at the Duke Medical Center; Dr. John A. Mahafee, professor and head of anesthesia at the Medical

College of South Carolina; Rudy Spears, president of the Spears Welding Supply Co., Greensboro; and Larry Dempsey, also of Greenshoro, eastern North Carolina representative for the Southern Oxygen Co.

* * *

Dr. Keith S. Grimson, professor of surgery at the Duke University Medical Center, has been elected vice president of the Society for Vascular Surgery.

Elections were held during the group's annual meeting just concluded in New York City. Dr. F. A. Simeone of Western Reserve University was chosen president of the Society, which numbers some 200 surgeons concerned with blood vessel and heart surgery.

Dr. Grimson, who joined the Duke Medical faculty in 1940, is a graduate of Rush Medical College of the University of Chicago. He is known for his research dealing with the use of drugs and special surgical procedures for relieving high blood pressure.

NEWS NOTES FROM THE BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

On May 19 and 20, investigators from throughout the United States attended a closed conference on urinary macromolecules at the Bowman Gray School of Medicine. It was the first conference on this subject to be held anywhere.

The purpose of the conference, supported by grants from the National Institute of Arthritis and Metabolic Diseases and the John A. Hartford Foundation, was to correlate, clarify and interpret findings from several disciplines; to compare methods of investigation; and to reveal areas which need concentrated study.

The 22 participants included Dr. Norman Boas of the Norwalk Hospital, Norwalk, Connecticut; Dr. Karl Schmid of the Massachusetts General Hospital, Boston, Massachusetts; Dr. J. A. Cifonelli of LaRabida University of the Chicago Institute, Chicago, Illinois; Dr. Zacharias Dische of Columbia University; Dr. Igor Tamm of the Rockefeller Institute; Drs. H. R. Catchpole, M. B. Engel, and Richard Winzler of the University of Illinois College of Medicine; and Dr. G. Berenson of Louisiana State University School of Medicine.

Presentations from the Bowman Gray School of Medicine were by Dr. William H. Boyce, professor and chairman of the Section on Urology; Dr. J. Stanton King, research assistant professor of biochemistry; and Mr. Marvel L. Fielden, research assistant in biochemistry and urology. Dr. J. Maxwell Little, professor of physiology and pharmacology, and Dr. Harold O. Goodman, assistant professor of medical genetics, were discussants of these presentations.

On May 26 and 27, the medical school was host to about 40 medical investigators from 13 institutions for meetings of the Actue Leukemia Cooperative Group B.

The group was organized in 1957 by the National Institutes of Health for the purpose of correlating the results of leukemia cases seen in each institution.

Dr. Charles L. Spurr, professor of medicine; Dr. Donald M. Hayes, assistant professor of medicine; and Dr. Richard B. Patterson, instructor in pediatrics (hematology), were participants from the Bowman Gray School of Medicine.

Outstanding investigators from the University of Miami School of Medicine, Emory University School of Medicine, the Medical College of Virginia, Walter Reed Army Hospital, the National Cancer Institute, the Children's Hospital of Philadelphia, Jefferson Medical College, Mt. Sinai Hospital at New York City, Maimonides Hospital at Brooklyn, Darmouth Medical School, Roswell Park Memorial Institute at Buffalo, and the Mayo Clinic attended the conference.

* * * * *

A medal honoring the late Dr. William Allan was inaugurated May 3 at a banquet of the American Society of Human Genetics at Atlantic City, New Jersey.

Ceremonies included the presentation of the first medal to Dr. Allan's widow, Mrs. Louisa P. Allan of Charlotte, and a review of the life and works of Dr. Allan by Dr. C. Nash Herndon, a former colleague of Dr. Allan and now professor and chairman of the Department of Preventive Medicine and Medical Genetics at the Bowman Gray School of Medicine.

The medal will be awarded annually "for outstanding research in human genetics." Dr. Allan organized the Department of Medical Genetics at the Bowman Gray School of Medicine in 1941, the first such department in the United States, and was professor of medical genetics here until his death in 1943.

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More than 100 college premedical students and their faculty advisors visited the medical school on Saturday, May 13, for a College Day Program.

Designed to familiarize prospective students with the details of and opportunities for careers in medicine, the program included tours of the medical school and N. C. Baptist Hospital and a luncheon in the hospital cafeteria. Dr. Donald M. Hayes, assistant dean, spoke to the students.

Students, primarily sophomores and juniors, came from Guilford College, N. C. State College, Davidson College, Woman's College, Catawba College, Elon College, East Carolina College, Lenoir Rhyne College, Furman University (South Carolina), Carson-Newman College (Tennessee), and Emory and Henry College (Virginia).

Dr. Frank R. Lock, professor and chairman of the Department of Obstetrics and Gynecology, was elected a vice president of the American College of Obstetricians and Gynecologists.

News Notes from the University of North Carolina School of Medicine

Exercises in honor of the graduating class were held by the University of North Carolina School of Medicine on Monday, June 5. Dr. W. Reece Berryhill, dean of the School of Medicine, presided.

The principal speaker was Dr. Louis G. Welt, professor of medicine. Also on the program was Richard W. Hudson of Vandemere, president of the graduating class.

Dr. Hugh McAllister, president of the UNC Alumni Association, presented the graduating seniors with certificates certifying membership in the Association.

A number of honors and awards for the year were announced during the exercises. Among them were the following: The American Psychiatric Association Student Thesis Award sponsored by Smith Kline & French to Clark Miller Hinkley of Waynesville; Bausch and Lomb Medal for Meritorious Student Research, American Society of Clinical Pathology to William Stephen Gibson, Jr., of Goldsboro; The Deborah C. Leary Memorial Award to William Mynell Clarke of Fayetteville; The Isaac H. Manning Award to Edward Arthur Sharpless of Chapel Hill; Mosby Book Awards to Robert DeVane Croom, III, of Maxton, James Lee Parker of Enfield, Carl Glenn Pickard, Jr., of Asheville' Benjamin Garrou of Valdese and Zell Allison McGee of Winston-Salem; and The Roche Award of Zebulon Weaver, III, of Asheville.

* * *

Three major annual awards to faculty members and members of the house staff of N. C. Memorial Hospital and students were announced recently by the School of Medicine at the annual Student-Faculty Day. Also announced were the new officers for the next school year of the Whitehead Medical Society.

The Professor Award, presented each year by the senior class of the Medical School, went to Dr. W. Geoffrey Wysor, Jr., of the Department of Medicine.

The William deB. MacNider Award went to Robert J. Cowan second year medical student of Greensboro.

The Henry C. Fordham Award was presented to Dr. William E. Easterling of Raleigh.

New officers who will head the Whitehead Medical Society next year are C. Glenn Pickard, Jr., Asheville, president; Fred Summers, Jr., Statesville, vice-president; Neil Bender, Pollocksville, secretary; and James Earnhardt, Thomasville, treasurer.

* * *

Plans for the transfer of $25,000 to the Department of Pediatrics of the University of North Carolina School of Medicine from Sigma Sigma Sigma Sorority, a national social organization, have been announced by Mrs. Dennis A. Kitchen of Kenmore, New York, and Dr. Floyd W. Denny Jr., chairman of the Department of Pediatrics.

The money will be used toward the completion of an isolation unit on the seventh floor of the hospital, which is the section of the hospital devoted to treating children.

* * *

Dr. Maxwell M. Wintrobe, head of the Department of Medicine of the University of Utah College of Medicine delivered the fifth annual Adam T. Thorp III Memorial Lecture at the University of North Carolina School of Medicine on May 24. He spoke on the subject of "Pebbles, Stones and Keystones."

* * *

New officers have been elected for the coming year by the Chapel Hill Chapter of the American Association of University Professors.

Dr. G. P. Manire, professor of bacteriology and immunology, was elected president.

* * *

Dr. George C. Ham, chairman of the Department of Psychiatry, was guest speaker at the Michael Reese Hospital Institute for Psychosomatic and Psychiatric Research and Training for their tenth anniversary celebration, held in Chicago recently. Dr. Ham soke on "Recent Trends in Psychiatric Education."

While in Chicago, Dr. Ham also delivered an address on "Psychoanalytic Education and the Psychiatric Residency" at the annual meeting of the Academy of Psychoanalysis and attended meetings of the American Psychoanalytic Association and American Psychiatric Association.

* * *

The Section of Physical Therapy of the University of North Carolina School of Medicine held exercises in decognition of four graduating seniors at the school on May 20.

The guest speakers was Dr. Catherine Worthingham, director of the Department of Professional Education of The National Foundation. Her topic was "The Dilemma of the Physical Therapy Profession."

The four graduates are Mrs. Elizabeth Brown Aman, Jacksonville; Mrs. Julianne Benson Smith, Lumberton; Miss Dorothy D. Hulick, Raleigh, and Kenneth Jordan of Asheboro. The degree of bachelor of science in physical theraupy was awarded these students at the regular graduation program of the University June 5. the University of North Carolina is the only school in the state offering a degree in physical therapy.

* * *

Dr. Robert R. Cadmus, director of N. C. Memorial Hospital, was a principal speaker at a meeting of Districts Four and Five of the N. C. Hospital Association held in Raleigh recently.

Joseph E. Barnes, director of Rex Hospital presided over the session. He spoke on "Physician-Hospital Relations."

* * *

Dr. Robert A. Ross, head of the Department of Obstetrics and Gynecology of the University of North Carolina School of Medicine was guest speaker at a meeting of the Virginia Academy of General Practice in Washington, D. C., on May 12. Dr. Ross discussed "Gynecological Aspects" on a symposium devoted to back pains.

* * *

Some 40 representatives from North Carolina's professional groups and state agencies resposible for the mentally retarded met in conference May 23-25 at N. C. Memorial Hospital.

The title of the conference was "Mental Retardation: Needs and Resources in North Carolina."

North Carolina Heart Association

The North Carolina Heart Association has set a deadline of August 1, 1961, for receiving applications for research grants-in-aid up to $2,000, except in unusual circumstances when they will consider applications for larger amounts from investigators within the state working in the cardiovascular field. These grants-in-aid are awarded by the Heart Association and its chapters to scientists who need interim or supplementary financial support for on-going projects, or who wish to demonstrate by a pilot experiment the value of a new project.

Awarded three times a year, the grants-in-aid are one phase of the Heart Association's research program, which is supported by public contributions to the annual Heart Fund campaign.

Applications for these grants may be forwarded to Dr. J. Logan Irvin, Chairman, Research Committee, North Carolina Heart Association, Miller Hall, Chapel Hill, North Carolina.

This research program is separate from that of the American Heart Association, which annually makes numerous research grants to scientists in North Carolina. Those interested in inquiring about the national program are asked to write to the American Heart Association, 44 East 23rd Street, New York 10, New York.

Association for Research in Ophthalmology

The Southeastern Section of the Association for Research in Ophthalmology will hold its next meeting at Duke University Medical Center January 12-13, 1962. All ethical physicians are invited to submit an abstract for this meeting prior to December 1, 1961. Address abstracts and inquiries to Dr. J. Lawton Smith, Department of Surgery, Duke University, Durham.

Edgecombe-Nash Medical Society

The regular meeting of The Nash-Edgecombe Medical Society was held in Rocky Mount on June 14.

Dr. John Chambliss presided over the showing of a 22 minute sound and color film, "External Cardiac Massage," which depicts the basic technique and value of closed chest resuscitation and shows procedures to be taken when heart arrest or ventricular fibrillation occurs in or out of the hospital.

News Notes

Dr. Charles M. Norfleet and Dr. Wayne E. Davis have announced their association for the practice of urology, with offices in the Professional Building, Cloverdale Avenue, Winston-Salem.

Dr. Charles T. Medlin has announced the association of Dr. Davey B. Stallings in general practice at Rural Hall.

AMERICAN COLLEGE OF GASTROENTEROLOGY

The American College of Gastroenterology announces that its annual course in postgraduate gastroenterology will be given at the Sheraton-Cleveland Hotel in Cleveland Ohio, on October 26-28, 1961.

The faculty will be drawn from the medical schools in and around Cleveland. The subject matter to be covered, from a medical as well as a surgical viewpoint, will be the advances in diagnosis and treatment of gastrointestinal diseases. There will be comprehensive discussions of pancreatic disease, biliary tract disease, electrolytes, peptic ulcer, etc.

There will be an "X-ray Classroom" on the last afternoon, presented by an outstanding panel of specialists who will answer questions and present instructional demonstration. There will also be a class in cinegastrophotography.

One complete sesion will be held at the Cleveland Clinic and one at the Cleveland Academy of Medicine.

For further information and enrollment write to the American College of Gastorenterology, 33 West 60th Street, New York 23, New York.

AMERICAN HEARING SOCIETY

Furthering its efforts to recruit teachers of lipreading, the American Hearing Society has awarded its 1961 Kenfield Memorial Scholarship to Miss Jean A. Mariast, of San Francisco. She plans to use the funds for special training at San Francisco State College.

Funds for the scholarship project were subscribed in 1937 in memory of Miss Coralie N. Kenfield, San Francisco, who was well known for her effective methods in teaching hard of hearing persons to lipread. The 1961 scholarship is the twenty-second award to be made by the society in 24 years, to the benefit of countless numbers of children and adults throughout the country.

AMERICAN PUBLIC HEALTH ASSOCIATION

Establishment of the annual Bronfman Prizes for Public Health Achievement, a new series of major awards to honor "outstanding current creative work leading directly to improved health for large numbers of people," was announced recently by the American Public Health Association. The prizes have been established with a grant from the Samuel Bronfman Foundation, Inc.

Dr. Berwyn F. Mattison, executive director of the Association, said that from one to three prizes will be awarded annually, depending on availability of suitable candidates. Each prize will consist of a $5,000 cash award and a commemorative symbol, now being devised. First awards will be announced and presented during the Association's eighty-ninth annual meeting in Detroit, November 13-17.

The Bronfman Prizes will be awarded for work of particular effectiveness in applying newer scientific knowledge to community health rather than for achievements in basic or laboratory research. With the establishment of the Bronfman Prizes, the Association this year continues to grant only one other award—the annual Sedgwick Memorial Medal in recognition of long and distinguished careers in public health.

U. S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE

Publication of the third edition of the annual directory and bibliography of the National Institutes of Health has been announced by the Public Health Service.

The publication is intended for reference use by research workers in the biomedical sciences inside and outside the federal government. It presents the broad outlines of NIH structure, names the professional staff, and lists more than 1600 scientific and technical papers deriving from the NIH program of direct research—laboratory and clinical—during 1960.

Designed to increase the usefulness of the volume are two indexes: one lists staff members and authors; the other—a subject index—reflects the scope of NIH research and provides quick reference to major and subordinate research areas. These areas covered the vast extent of problems that concern biomedical scientists—from the biochemistry within the human cell to the causes and control of the degenerative diseases, and the physiological and psychological aspects of aging.

Copies of the publication are available from the Public Information Section, Office of Research Information, National Institutes of Health, Bethesda, Maryland.

* * *

About 1,161,000 of the civilian, noninstitutional population of the country have hearing aids, according to a new report from the Public Health Service's National Health Survey. This total represents one-fifth of the people who are reported to have hearing impairments.

The estimates are derived from household interviews conducted during the year ending June 30, 1959, with a representative sample of the population.

About 253,000 people had wheel chairs during this period, the new report shows. Some 54 per cent of these were persons so disabled as to be confined to the house except in emergencies.

Estimates of 201,000 persons with leg or foot braces and 494,000 other types of braces were reported by the survey. About 82,000, or 41 per cent of the leg or foot braces, were for children under 15 years of age, and about three-fourths of these 82,000 braces were worn because of conditions due to poliomyelitis or of congenital origin.

Of the estimated 139,000 people with artificial limbs, 132,000, or 94 per cent were males. About 106,000, or 76 per cent of the total number, were persons with an artificial leg or foot.

The report is entitled, "Distribution and Use of Hearing Aids, Wheel Chairs, Braces, and Artificial Limbs, United States, July 1958—June 1959," Public Health Service Publication No. 584-B 27. Copies may be obtained from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., at 25 cents a copy.

* * *

The Public Health Service recently announced release of a new publication which reveals serious health hazards in uranium mining and urges more aggressive action to meet the danger. The report summarizes the proceedings of the Governors' conference on health hazards in uranium mines held last December, a meeting attended by officials from the seven Western states engaged in uranium mining. In spotlighting the problems of uranium miners, the conference revealed that, among these workers, deaths are occurring in excessive numbers due to lung cancer and to complications of silicosis.

The report concludes that mounting evidence of the severe health hazards in uranium mines points out the urgency for more aggressive action to meet the problem. Control of these hazards was viewed by the Conference participants as a primary responsibility of the states. Upon request, various agencies of the Federal Government stand ready to provide assistance to the states in carrying out this responsibility.

* * *

Scientists of the Public Health Service's National Cancer Institute have developed a simple, rapid method of drying cells for microscopic study. The new method permits indefinite storage of dried cells and eliminates chemical treatment, which may disturb vital details of cell structure and function.

A report of the work was presented recently at the Syverton Memorial Symposium and the twelfth annual meeting of the Tissue Culture Association held at Detroit, Michigan. The report was prepared by Henry C. Orr, Dr. Morris Belkin, and Walter G. Hardy, all of the National Cancer Institute, and a former coleague, Dr. Ezio Merler, now of the Harvard Medical School, Boston, Massachusetts.

* * *

The Chemotherapy Service of the National Cancer Institute is initiating studies on the chemotherapy of medulloblastoma with regional antimetabolites at the Clinical Center, National Institutes of Health, Bethesda, Maryland.

A limited number of patients who have undergone neurosurgery and radio-therapy will be accepted for study. Referrals of such patients will be greatly appreciated.

Physicians who wish to have their patients considered for this study may write or telephone:

Dr. Myron Karon
Chemotherapy Service
General Medicine Branch
National Cancer Institute
Bethesda 14, Maryland

VETERANS ADMINISTRATION

A new radioactive agent has been developed by Veterans Administration research for use in the diagnosis of kidney disease.

Called radioactive hippuran, it is used to differentiate between high blood pressure caused by kidney disease, which may be surgically curable, and the more common type of high blood pressure of unknown cause for which there is no known cure.

Radioactive hippuran will soon be employed in a nationwide survey of patients with high blood pressure. This survey is being supported by the United States Public Helath Service which has granted $100,000 to the National Institutes of Health to conduct the two-year long program.

* * *

Persons with severe mental illness (psychosis) are apparently free of a number of common physical ailments, a 15-year survey of Veterans Administration patients shows.

Among hundreds of mentally ill veterans studied at the Bedford, Massachusetts, VA hospital over the past decade and a half, not a single case of hay fever was discovered and only one case of asthma was found. Rheumatoid arthritis was rare.

In addition, many mental patients may not show signs of discomfort or pain during major painful physical disorders.

The investigation reveals that most neuropsychiatric patients do not complain of physical pain even with perforation of peptic ulcers, acute appendicitis, and severe heart attacks.

Dr. Walter E. Marchand, chief of the Bedford VA hospital's medical-surgical service, began the study of unusual aspects of the practice of medicine among psychotics in 1945. In 1952 he was joined by Dr. Otto F. Ehrentheil, clinical instructor in medicine at Tufts University School of Medicine.

It shows that psychotic patients brought to surgery for acute peritonitis often had suffered the perforation 48 to 72 hours earlier. Autopsy reports disclose that some patients had suffered heart attacks several days before their death and had shown no signs of physical pain.

Thus, the study urges, physicians, nurses, and psychiatric aides should be aware of any slight change in the patient's behavior or lapse from his usual routine.

Dr. Ehrentheil and Dr. Marchand have summed up their findings in a book, "Clinical Medicine and the Psychotic Patient." Written in an informal narrative fashion, it abounds in observations and practical advice of value not only to psychiatrists but to any physician who undertakes the care of a psychotic patient.

Dr. Gasque Appointed Medical Director of Chemical Company

Dr. Mac Roy Gasque has been appointed to the newly created post of medical director of Olin Mathieson Chemical Corporation, Gordon Grand, vice president-law and administration, announced. Dr. Gasque had been medical director of Olin's Ecusta and film operations at Pisgah Forest, North Carolina.

As corporate medical director, Dr. Gasque will develop and implement modern industrial health programs for the corporation which emphasize preventive medicine and health promotion among employees. These programs, to be coordinated with Olin's safety program, will be tailored to meet individual plant and community needs.

Dr. Gasque holds a faculty appointment in the department of preventive medicine at the Duke University Medical School and is a frequent lecturer at the Bowman Gray School of Medicine and the University of North Carolina. His principal office will be located at 460 Park Avenue, New York City.

Maytag Gives Grant to NFME

A $2,000 grant to the National Fund for Medical Education, which gives financial aid to the nation's 85 medical colleges, has been announced by Robert E. Vance, president of the Maytag Company Foundation, Inc.

Other grants announced simultaneously went to the Iowa College foundation and the United Negro College fund.

Each of these groups has received grants annually since establishment of the Maytag foundation in 1953. The foundation receives its financial support from the Maytag company, home laundry appliance manufacturer.

Attack on Counterfeit Drugs Proposed

A four-prong attack on prescription drug counterfeiting was proposed recently by a representative of one of the nation's leading ethical drug manufacturing firms.

George H. Hambrecht, of the legal department of Smith Kline & French Laboratories of Philadelphia, said before the eightieth annual con-

JAMA
THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION
VOL. 174, NO. 5 OCTOBER 1, 1960

newest
J.A.M.A.
paper[1]
reports

DBI

"oral therapy of choice"
in management of diabetes . . . from the
mild stable adult to the severe labile juvenile

vention of Indiana pharmacists meeting last month that "seizures of counterfeit drugs show there is still an area of irresponsibility in which this vicious racket operates."

Hambrecht outlined this program to attack drug counterfeiting:

1. Legal action by reputable manufacturers against "fly-by-night" operators engaged in counterfeiting operations, coupled with education programs by the industry to alert retail pharmacists to counterfeiting operations.
2. Effective enforcement of state statutes outlawing counterfeiting.
3. Action through the Federal Food, Drug and Cosmetic Act.
4. Action by state pharmacy boards against individual retailers dealing in counterfeits.

New Device Speeds Relief from Migraine N

"Almost immediate" relief from severe migraine, with some patients reporting relief in five minutes and the average requiring ten minutes, was reported on June 25, with the use of a new device for rectal instillation of medication.

Dr. Robert E. Ryan of St. Louis, Mo., reported to the American Association for the Study of Headache, meeting in the Park Sheraton Hotel, New York, that the quick results had been obtained by eliminating the semi-solid base of suppositories, which must melt before drugs can enter the blood stream.

The doctor reported on the use of Rectalad Migraine (Wampole Laboratories), a plastic tube containing four anti-headache drugs in liquid solution. The 2 cc. dose contains the drugs in combination determined most effective through experiment: 2.0 mg. ergotamine tartrate, 25.0 mg. caffeine, 0.4 mg. scopolamine aminoxide, and 200.0 mg. chloral hydrate.

Among 200 patients, 168 obtained complete relief and 16 obtained partial relief, while the remaining 16 apparently were not helped. Side effects consisting of cramps in 10 cases and mild laxative effect in 3, were minimal. There was no incidence of vomiting.

Rr. Ryan noted that Rectalad Migraine eliminates a major disadvantage of suppositories: a tendency to melt in summer. But the chief advantage, he said, was speed of relief.

The plastic tube dosage form, in which the drugs are suspended in a nonirritating liquid vehicle to minimize laxative effect, may be self-administered.

Indications that schizophrenia, this nation's number one mental disorder, runs in families are cited by the March Issue of **Patterns of Disease**, a monthly Parke, Davis & Company publication.

results of 104 "problem" diabetics treated with...

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stable adult diabetes • sulfonylurea failures
unstable (brittle) diabetes

1. Barclay, P. L.: J.A.M.A. 174:474, Oct. 1, 1960.

NOTE – before prescribing DBI the physician should be thoroughly familiar with general directions for its use, indications, dosage, possible side effects, precautions and contraindications, etc. Write for complete detailed literature.

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The Month in Washington

The American Medical Association supported the Kennedy Administration's proposal to provide $750 million in matching funds for construction of medical, dental, public health, and osteopathic schools.

In a letter to Senator Lister Hill (D., Ala.), chairman of the Senate Labor and Public Welfare Committee, Dr. F. J. L. Blasingame, executive vice president of the A.M.A., said:

"As an Association of 179,000 practicing physicians, we are vitally interested in maintaining the high quality of medical education in the United tSates because of its direct relationship to medical care. For over a century, the American Medical Association has been actively and effectively engaged in the improvement of medical education in the United States. It can now be said, with assurance, that medical education in this country is superior to that found anywhere else in the world. It is not a coincidence that the improved standards of medical care in the last half century saw the elimination of sub-standard medical schools and diploma mills which had been turning out graduates in large numbers. This improvement in medical education is the result of the vigorous efforts of this Association and other interested organizations.

"We strongly believe that increased attention must be given to the adequacy of physical facilities, the availability of qualified instructors and the availability of teaching material and patients for the clinical phases of medical education if high standards of medical education are to be maintained. Any attempt to increase the number of medical students without regard to these conditions will result in a lowering of the standard of medical education. We are of the firm conviction that increase in the physical facilities available for medical education should be given priority at this time over any other federal legislation in the field of medical education.

"We believe that there is need for assistance in the expansion, construction and remodeling of the physical facilities of medical schools and, therefore, a one-time expenditure of federal funds on a matching basis, where maximum fredom of the school from federal control is assured, is justified."

The A.M.A. opposed a provision that might encourage medical schools to expand too rapidly. Dr. Blasingame said: "It is quite possible that a forced increase in freshman enrollment would be detrimental to the quality of medical education."

The Association did not take a position on the provision of the Administration legislation that would provide federal scholarships to medical students. However, Dr. Blasingame described to the senate committee A.M.A.'s new medical scholarship and student loan programs.

The General Accounting Office found the Defense Department's Medicare program being conducted generally "in a satisfactory manner," but recommended some changes designed to correct what it considered "important deficiencies."

The Army, which administers the program of medical care for dependents of members of the armed services, took steps to put into effect most of the recommendations of the GAO, which audits federal spending for Congress.

However, Medicare officials rejected a GAO proposal for a change in physician fee.

"Our view disclosed that physicians' claims for medical care are, in general, significantly higher in states where maximum fees are made known to physicians than in those states where maximum fees are not made known," the GAO reported. "We estimate that there is an additional cost of as much as $3 million to $4 million annually as a result of maximum fees, rather than normal fees, being charged in the states where fee schedules are distributed to the physicians."

The GAO recommended that lower fixed fee schedules be negotiated for states where a high percentage of physicians claims are for maximum allowable fees, "subject to being raised only on the basis of clearly supported evidence of higher normal fees."

If lower fees cannot be negotiated, the GAO said, efforts should be made "to have

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From the Washington Office of the American Medical Association.

the state medical society or other appropriate parties accept the responsibility for determining that physician claims are generally not in excess of their normal charges."

The GAO further recommended that "physicians be required to certify on each claim that the amount billed does not exceed the physician's normal fee for the medical care furnished."

The Army disagreed, saying that it believed "the present contracting concept is the most suitable to meet the requirements and is in the best interests of the government."

The A.M.A. noted that it had held from the outset that "fixed fee schedules would result in a more expensive program than if physicians were permitted to charge their normal fees."

Fixed fee schedules call for some fees above some so-called normal fees and others below average fees, the A.M.A. said. "Psysicians tend to 'balance out' by using fees listed in the fixed fee schedule."

Medicare was started December 7, 1960. During the first four years of the program, $130 million was paid to civilian doctors and $133 million to civilian hospitals for care of 1.1 million military dependents. Maternity cases accounted for about half the total.

Medicare has asked Congress for $73.2 million for the fiscal year 1962, beginning this July 1. This is a $6.9 million increase over Medicare's current budget. The increase is needed, Medicare said, because of more military dependents eligible for the program's benefits and increases in the costs of services.

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Book Reviews

Myxedema. By Jerry K. Aikawa, M.D. 106 pages. Price, $5.00. Springfield, Ill.: Charles C Thomas, Publisher, 1961.

In a recent review of two books Mr. Sidney Harris commented that they both had a severe case of literary edema—"That disease so pandemic in the book publishing business." Dr. Aikawa's book is refreshingly free from this disease. He has put into 99 pages—including references—a fascinating history of myxedema, beginning with T. W. King's recognition of the function of the thyroid gland in 1836. Sir William Gull in 1875 reported five cases of myxedema and is credited with being the first to recognize it. Dr. William Ord, in 1878, described the pathology of the condition and first proposed the name **myxedema.** Brief sketches of Drs. Gull and Ord add interest to the story.

Chapter II describes the evolution of specific therapy, beginning with transplantation of the thyroid gland. Credit is given Dr. George Murray for first using, in 1891, a thyroid extract in a myxedematous patient, who continued to take the extract until she died in 1919 at the age of 74. Soon after Dr. Murray reported using the extract by hypodermic injection, three physicians reported independently that thyroid extract was just as effective by mouth as by hypodermic injection.

In Memoriam

Warren Edwin Miller, M.D.

Warren Edwin Miller was born January 22, 1092, in Garnersville, Georgia. He attended Davidson College and was graduated from Emory University in Atlanta, Georgia, where he received his M.D. degree in 1929. He served a one-year rotating internship at Grady Memorial Hospital in Atlanta and a two-year surgical residency at Union Memorial Hospital in Baltimore.

He then went to the Canado Mission on the Navajo Indian Reservation in Canado, Arizona, working with Dr. Salsbury and the Navajo Indians for two years. He came to Whiteville in 1935, and founded a small hospital known as Columbus County Hospital, which he operated for several years.

He was elected a Fellow of the American College of Surgeons in 1937.

He served for five years as chairman of the Selective Service Board, and in May, 1943, he volunteered and entered the U. S. Army Air Force with the rank of major. He served both in the States and in the Southwest Pacific. He was separated from the service in 1945 with the rank of lieutenant colonel, and resumed private practice in Whiteville.

Always interested in cancer, he became director of the North Carolina division of the American Cancer Society and was instrumental in the establishment of the Cancer Center for Incurables at Lumberton, North Carolina. At the time of his death he was president of the local chapter of the American Cancer Society.

Dr. Miller's love of flying was both for pleasure and for the benefit of his patients. Before the days of rapid pathologic reports he would fly to Wilmington with the specimens for early report. He has transported patients in his private plans to their homes after their initial stay in the local hospital and to medical centers for treatment. He was a charter member of the Flying Physicians Association and was cited by the United States government in June. 1959. for a successful flight to Alaska by the Flying Physicians to prove an evacuation plan.

Dr. Miller was married in 1936 to the former Elma Webster of Hamilton, Ontario. Two sons were born of this marriage.

Dr. Miller died December 10. 1960, in Columbus County Hospital at the age of 56, a victim of cancer.

Dow Names Dr. M. B. Chenoweth Research Scientist

Dr. Maynard B. Chenoweth, an internationally known pharmacologist on the Biochemical Research Laboratory staff of the Dow Chemical Company, has been named to the rank of research scientist.

Dr. Chenoweth is the eighth Dow scientific worker to be named to this classification, which accords recognition for outstanding achievements in science. His appointment was announced by Dr. R. H. Boundy, Dow vice president and director of research.

While his research has been over a wide range, Dr. Chenoweth is particularly known for his classical work on fluoroacetate and as an authority on metal binding in biological systems. More recently he has made major contributions to the field of anesthesiology.

Brantley Appointed District Manager By Wyeth Laboratories

The appointment of Charles H. Brantley, Raleigh, North Carolina, to the position of district manager with Wyeth Laboratories, has been announced by Stuart V. Smith, vice president and director of sales.

Brantley, who will have his headquarters in Raleigh, has been a member of the Wyeth sales staff since 1950. He is a graduate of Wake Forest College, and served with the Army Medical Department.

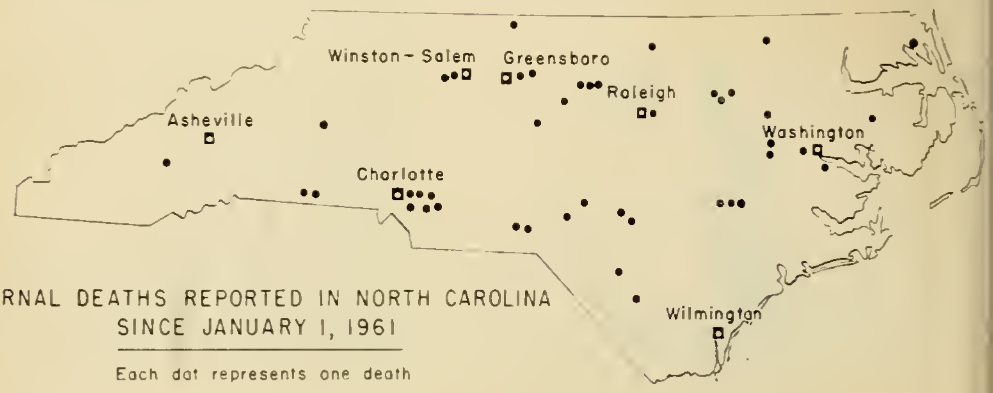
Brantley resides at 621 Powell Drive with his wife and four children.

New Dressing Announced by Johnson & Johnson

A new dressing which marks a milestone in the control of bleeding in both external and internal wounds is now available to the medical profession, it was announced by Johnson & Johnson, the world's largest manufacturers of surgical dressings. The new material. which perform its hemostatic function by inducing an artificial clot, is known as Surgicel.

Surgicel is applied in gauze-like strips or carded fiber pads which in appearance are not unlike absorbent cotton. Within two to three minutes after contact with the wound. bleeding stops and oozing is brought under control, even though the area may involve tissues which are characteristically insusceptible of suturing or ligaturing, or in which such procedures are difficult.

The extensive clinical tests, which included use of Surgicel in operations on the brain, were conducted principally by Dr. Miller at Fort Howard, Dr. Franklin at Jefferson Medical College; and by Dr. Elliott S. Hurwitt, chief of the surgical division at Montefiore Hospital and clinical professor of surgery at Columbia College of Medicine; and by Dr. R. W. Postlethwait, chief of the surgical division at the Veterans Administration hospital, Durham, North Carolina, and professor of surgery at Duke University, in addition to many other studies carried out in leading medical institutions here and abroad.



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IN THIS ISSUE:

Multiple Severe Automotive Injuries

George B. Johnson, M. D.

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VOLUME 22 AUGUST, 1961 No. 8

Multiple Severe Automotive Injuries

EVALUATION AND MANAGEMENT

GEORGE B. JOHNSTON, M.D., F.A.C.S.
ASHEBORO

From January 1, 1951 through December 31, 1960, there were 264,065 reported automotive accidents on the primary and secondary road system in North Carolina, causng 115,617 persons to be injured and 9,428 o be killed[1]. During this same period of ime in my home county of Randolph, located in the geographic center of the state, here were 6,945 traffic accidents, causing 2,356 persons to be bodily injured and 175 killed (table 1).

A great majority of these injuries were reated at Randolph Hospital in Asheboro, 100-bed general hospital. Most of the paients were treated in the emergency room nd allowed to go home, but 250 were classified as seriously injured. This number included both single and multiple injuries. Patients with single injuries had one preominant area of injury with one or more reas of lesser injury. Eighty-three persons ad severe, multiple injuries involving more han one body system and/or more than ne extremity injury. Of these 83 patients 3 had injuries considered to be mutilating n type. Eighteen expired less than 24 hours fter admission. In 14 patients the primary ause of death was severe craniocerebral inury; in 1, cardiac contusion and "wet" lung yndrome; in 2, retroperitoneal hemorrhage om torn kidney pedicle; and in 1, cornary occlusion, probably occurring before e accident.

Twelve patients died after being hospitalized more than 24 hours. Craniocerebral injury was the primary cause of death in 4; lower nephron syndrome in 3; probable fatty embolus in 2; overwhelming sepsis and uremia in 1; retroperitoneal and intra- abdominal hemorrhage from torn kidney pedicle in 1; a rapidly advancing venacaval thrombosis and bronchial pneumonia in 1. Twenty-three of those with mutilating injuries and 30 with less severe multiple injuries, were discharged ambulatory (table 2). The most frequent combination of injuries in our group of cases was that of craniocerebral and extremity long-bone injuries. Next frequent was thoracic and extremity injuries. Almost all of our severely injured patients had some degree of craniocerebral injury. In some cases, however, it was not the primary injury.

General Observations

There are no reliable stastistics which show the actual number or percentage of multiple injuries, but the impression is that they are becoming more common. In the investigation of automotive crash injuries at Cornell University Medical College, 1,000 injury-producing accidents in which 1,678 occupants were injured were analyzed[2]. "Multiple injuries constitute the most common pattern; actually, over 66 per cent of all injured persons suffered injuries to two or more bodily areas."

Despite the remarkable advance of medicine since the beginning of the century, there is one field in which progress has been woefully slow—the care of the injur-

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Read before the Section of Orthopaedics and Traumatgy, Medical Society of the State of North Carolina, heville, May 9, 1961.

Table 1
Accidents Occurring on Primary and Secondary Road System in North Carolina
January 1, 1951 - December 31, 1960

	N. C.	Randolph Co.
Number of Accidents	264,065	6,945
Persons injured	115,617	2,356
Deaths	9,428	175

ed. One reason is that we are unable to evaluate the degree of malfunction of each body system in an individual who has sustained severe multiple injuries. The combinations of various degrees of injuries to different areas and systems could continue almost *ad infinitum*.

Another reason for the lack of progress in this field is that automotive injuries interest only a few physicians. Great care is given to the patient with a cardiac defect who has had or is going to have open heart surgery. But the same teen-ager who comes to the emergency room at 2:00 A.M. after a "drag session" on the highway, and who may have had a few beers "to give him courage, is not afforded the same devotion or painstaking study. Since many accident victims are service patients, the burden of care usually falls to the resident staff, and in many instances this is probably fortunate. The need for proper care constantly increases in this age of the machine. In 1958 an evaluation of emergency room care in this country was undertaken by Cornell University Medical College, under the direction of Dr. Preston A. Wade, professor of clinical surgery and then chairman of the American College of Surgeons Committee on Trauma. This committee became cosponsor of the project along with the American Hospital Association and the U. S. Public Health Service[3].

The purpose of the project was to evaluate the status of emergency care throughout the country and, from the data obtained, to develop a standard for the organization of emergency facilities and services. We are seeing the evolution of the old "accident room" into an emergency department which may become the medical center of the future, whether we like it or not. We must be certain that our emergency facilities are adequate to accept the responsibilities which the public is placing upo us[4].

Table 2
Automotive Injuries Treated at Randolph Hospital
January 1, 1951 - December 31, 1960

	No. Case
Single and multiple severe injuries	250
Severe multiple injuries	83
Mutilating injuries	53
Expired 24 hours or less after hospitalization	18
Expired after 24 hours hospitalization	12
Discharged ambulatory	23
Total number with multiple injuries discharged ambulatory	53

Preliminary Evaluation and Managemen

Except for an occasional consultatio with an out of town neurosurgeon, our pa tients were handled from their entrance in to the emergency room until their demis or discharge from the hospital by the ge eral practitioner and general surgeon o call at the time, who acted as staff physi ian, resident, intern, and occasionally a nurse and orderly.

We found it extremely difficult to cla sify these multiple injuries accurately a cording to severity in order to institu planned therapy. There seems to be no we documented formula by which one can pi eon-hole each patient and treat him accor ing to some preconceived plan which tested, tried, and true.

In evaluating an injured patient, it helpful to know as much as possible abo the accident. How fast was the car goin Was the patient thrown out? Was he in th front or back seat? Was he driving?

Our most seriously injured patients a those who were thrown out of the vehic and who usually sustained head injuri and mangled extremities. The driver is u ually the less seriously injured passenge but he is the one most likely to susta cardiac contusion.

It is also important to get a history the patient's state of consciousness sin the accident, keeping in mind the "laten period of epidural hemorrhage. With spin injuries causing paralysis, it is important know if the paralysis was immediate or d layed. If relatives are present—which they usually are—it is helpful to determine the history of any disease such as diabetes, heart disease, or allergy that may complicate treatment. For one reason or another, we are rarely able to elicit all the needed information. Usually the patient is brought to us, unconscious, at the emergency room with the history that he was in a "bad wreck" and was thrown from or pinned under the car, as the case may be. This helps, but it leaves a great deal to be desired as far as history is concerned.

In the management of the injured patient, respiratory distress has the highest priority for attention and treatment. In our experience, it is most frequently caused by a flail chest or open thoracotomy. If the airway is obstructed by vomitus or impacted dentures, a tracheostomy may be life saving.

Next in priority is severe external hemorrhage. The site should be located immediately and controlled with hemostats or pressure dressings as indicated. Other measures should be well coordinated, and several life saving procedures can be done simulatneously by different members of the emergency team. The degree of shock should be evaluated early and appropriate measures instituted immediately. If extensive fractures or hemorrhage is present, blood should be cross-matched and administered as soon as possible. Dextran or some other suitable plasma expander may be started at once. Levophed (norepinephrine) has been found to be life saving in many instances until blood transfusions can be started.

There seems to be no single, commonly observed clinical sign of shock that is reliable. Its manifestations vary. Experience has taught that certain types of injury produce shock. In the presence of these injuries, even when the usual low blood pressure or weak, rapid pulse are absent, it is wise to institute shock therapy. In an effort to keep shock at a minimum, the patient is handled and moved as little as possible. Usually, in our institution, all emergency treatment is given and x-rays are made while the patient is on the ambulance stretcher, and frequently he is taken to the operating room on the same stretcher. Only those x-rays which are absolutely necessary *at the time* are done. Sometimes this rule is made difficult by demanding relatives who seem to think that an x-ray picture is a therapeutic rather than a diagnostic procedure.

Detailed Examination and Treatment

All clothing is removed, usually by cutting. The head is palpated and examined for blood or cerebral spinal fluid exuding from the orifices. The head is palpated for depressions, lacerations, and contusions. The cervical spine is examined and any tenderness or obvious deformity noted. The examination extends distally. The clavicles and chest are palpated for fractures, and the type of respiratory excursions is observed. The abdomen is examined for contusions, discolorations, tenderness, and rigidity. Pressure is made over the pubis and iliac crest. The extremities are then noted for deformities or limitation of motion.

Head and thorax

In the evaluation of head injury, the degree of unconsciousness is of primary importance. The changing picture of the state of consciousness, pulse, temperature, blood pressure is noted, as are the equality of the pupils, movement of the extremities, and depression of the deep tendon reflexes. Signs of localization of injury from subdural or epidural hematoma indicate a grave prognosis, and trephination may be a life saving procedure. It has been our experience, however, that actual localization of brain injury from automobile accidents has been the exception rather than the rule. Usually, there is a vast brain contusion or laceration rather than a single localized hematoma. We do not elevate depressed fractures in silent areas of the brain immediately, but wait until more pressing problems are met. Open skull fractures and depressed fractures over dural sinuses and nonsilent areas of the brain have a high priority of operative treatment.

We next evaluate the chest, noting the quality of respiratory excursions, subcutaneous emphysema, and paradoxical movements of the flail chest. Fractured ribs are noted for point tenderness and pain is reduced by intercostal nerve block. In the presence of subcutaneous emphysema, tension pneumothorax should be guarded against. Needle aspiration of the chest may determine the need for underwater, intercostal drainage. The demonstration of a flail chest usually necessitates the use of towel clips attached to the rib cage and suspended by overhead weight.

Abdomen

Intra-abdominal injuries are sometimes difficult to evaluate. In some cases the usual signs of massive injuries are lacking or overlooked because of shock, prostration, from more painful injuries elsewhere. Confusion may also occur during the early stage of an injury from the effort to differentiate intra-abdominal injury from abdominal wall injury. An upright roentgenogram of the diaphragm for free air will be helpful in determing the presence of a perforated hollow viscus.

Signs of increasing shock and increasing, non-shifting dullness, and bulging of the left flank may indicate splenic rupture. An extremely helpful diagnostic procedure is needle apsiration of the abdominal cavity.

Every severely injured patient should be catheterized and the urine examined. Usually, gross blood means urethral or bladder injury. Microscopic evidence of blood may mean kidney damage. If injury of the bladder is suspected, sterile saline is instilled into it and the amount of fluid returned is measured. If the amount is less, a leak in the bladder is suspected. Failure to pass the catheter usually signifies a torn urethra, and a suprapubic cystostomy is indicated. Microscopic blood is frequently found in the urine specimen of accident victims with severe multiple injuries.

In order to evaluate the urinary tract more completely, an intravenous urogram and a cystogram are made when conditions permit, or when they seem indicated. Severe hemorrhage from a torn kidney pedicle is usually manifested by the presence of almost irreversible shock, and immediate operation is indicated as a life saving procedure. Frequently it is too late. Many cases of kidney fracture may be treated without operation.

Extremities

We believe that injuries of the extremities present less of an emergency than internal ones. Obvious fractures do not require immediate x-ray studies or reduction. We believed that open fractures should be cleansed and debrided and splinted in the emergency room, then left open for operative reduction at a more favorable time. In the patient with severe multiple injuries, we do not reduce even the closed fractures until his condition has become stabilized.

Major injury to soft tissue is also less urgent than the previously menioned injuries. They are usually cleansed and debrided gently at the time of admission to the emergency room and closed at a more appropriate time. Minor lacerations can be closed in the emergency room under local anesthesia.

Conclusion

An ever increasing number of automotive injuries are being treated in our hospitals each year. Among them are a growing number of multiple injuries of almost infinte variety, each testing the judgment of the attending physician to the limit. It is thought that these injuries can be handled best under the direction of a single wel trained physician who is able to meet the demands of thoroughness and common sense, and who can make sometimes bold decisions rapidly and execute them efficiently.

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Survey of Poliomyelitis in North Carolina, 1959

ROBERT W. DARTER, M.D.[*]
PHOENIX, ARIZONA
and
JACOB KOOMEN, M.D. M.P.H.[†]
RALEIGH

During 1959 the incidence of paralytic poliomyelitis in North Carolina increased sharply in contrast to the consistent decline observed each year since 1955. A similar increase in incidence was observed generally throughout the United States. Observations made in North Carolina during 1959 were compared with data from other years to determine possible reasons for the increase. This report provides comparative statistics on the disease for the past 20 years, and considers differences in the distribution by age and race during 1959 in contrast to that of previous years.

In the years immediately before extensive use of the killed virus vaccine, poliomyelitis was reported primarily in white school-age children distributed rather uniformly throughout all socioeconomic groups[1]. Subsequent to the widespread use of vaccine, different trends in the epidemiology of poliomyelitis have become apparent in the United States. In several localized epidemics since 1955, a shift in age and race incidence has been demonstrated[2]. Statistics disclosed that cases were observed predominantly in children of preschool age and that attack rates among Negroes equalled or exceeded those among whites. The shift in age incidence was also manifest in the national statistics compiled by the Poliomyelitis Surveillance Unit of the Communicable Disease Center[3].

Methods

Data pertaining to poliomyelitis morbidity in North Carolina have been collected during the past 20 years by means of a report card system. The physician making the diagnosis submits a morbidity report to the health department in the county of the patient's residence. The health department forwards the card to the Communicable Disease Control Section of the State Board of Health. The data are checked for completeness, and any deficiencies are brought to the attention of the reporting health unit. Ultimately the information is transferred to IBM punch cards from which the statistics for cases reported prior to 1955 were compiled.

Prior to 1955 paralytic and nonparalytic cases were either not distinguished or were incompletely so. For this reason, all data presented for years prior to 1955 are for total cases of poliomyelitis.

Beginning in 1955 an intensive poliomyelitis surveillance program was initiated in cooperation with the Communicable Disease Center. Each reported case of poliomyelitis was also reported to the Poliomyelitis Surveillance Case Records. Data required to complete each record were obtained, so that information about immunization status, paralytic status, and 60-day follow-up is accurate and complete.

In addition, reports of suspected cases have been secured following the receipt of laboratory results from the State Laboratory of Hygiene and the virus laboratory of the Department of Pediatrics of the University of North Carolina Medical School. Death certificates were screened as to poliomyelitis. Occasional cases seen in orthopedic or crippled childrens' clinics for residual defects were reported. Close liaison has been maintained with the State Division of the National Foundation, and copies of requests for financial aid were forwarded to the CDCS by the National Foundation.

From the Communicable Disease Center, Public Health Service, U. S. Department of Health, Education, and Welfare, Atlanta 22, Georgia.

Prepared for presentation at the Annual Conference of the American Public Health Association, San Franisco, California, October 31-November 4, 1960.

[*] Epidemic Intelligence Service Officer, CDC, assigned to the Phoenix, Arizona, Field Station.

[†] Chief, Communicable Diseases Section, Division of Epidemiology, North Carolina State Board of Health, Raleigh, North Carolina.

Table 1
Age-And Race-Specific Attack Rates Per 100,000 Population For Poliomyelitis in 1940, 1948, and 1953 and Paralytic Poliomyelitis In 1955, 1957, and 1959, North Carolina

Year	No. Cases									
	Age and Race									
	0-4		5-9		10-14		15-39		Total	
	W	NW	W	NW	W	NW	W	NW	W	W
1940	35	8	14	2	10	2	2	1	61	13
1948	984	155	619	68	303	33	197	26	2,103	282
1953	313	43	247	18	140	8	145	8	845	77
1955	62	15	41	4	14	1	43	3	160	23
1957	12	16	4	3	3	1	6	2	25	22
1959	82	69	30	22	9	16	35	12	156	119

Year	Rates									
	Age Group and Race									
	0-4		5-9		10-14		15-39		Total	
	W	NW	W	NW	W	NW	W	NW	W	W
1940	14	6.9	5.3	1.7	3.6	1.6	0.2	0.2	2.4	1.3
1948	295	104	213	54	115	28	25	4.9	76.2	28.6
1953	87	27	80	13	51	6.7	12	1.8	20.8	6.8
1955	16	9.4	13	2.9	4.6	1.2	3.3	0.7	4.8	2.1
1957	2.9	9.9	2.1	2.2	1.0	0.8	0.4	0.4	0.9	1.9
1959	21	42	9.0	16	3.1	13	2.5	2.7	4.7	10.4

Because of the precise data available since 1955, statistics covering this interval are expressed in terms of paralytic cases only.

For the calculation of rates, yearly population estimates made by the Statistics Section of the State Board of Health were employed. Between 1940 and 1950 the yearly change was calculated. Since 1950 a straight line projection of the 1940-1950 change has been used. The age-specific population estimates were similarly made.

In tabulating the immunization status of the cases reported in 1959, it was decided that individuals receiving their first dose of killed virus vaccine within 10 days of onset would be considered unimmunized, since the inoculation would have been given during the probable incubation period of the disease and would not have contributed to their immunity. Eight persons with paralytic disease fell in this category. Only three persons received second doses of the vaccine within 10 days of onset. Since persons with one or two doses of vaccine are grouped together, no distinction regardin immunization status was made.

Observations and Discussion

Until the epidemic year, 1944, the ag distribution of poliomyelitis in North Carolina tended to resemble that of classic infantile paralysis. Half or more of the case reported up to this time occurred in children under 5 years of age. Beginning i 1944 an increasing proportion of cases occurred in school age children and youn adults. This trend paralleled that of th United States as a whole. Attack rates in th 0-4 and 5-9 age-group were approximatel equal between 1944 and 1954.

Between 1940 and 1954 the proportion o cases in Negroes did not exceed 20 per cen of the total reported cases, nor was the attack rate in Negroes ever as high as tha in whites for any age-group.

Following extensive use of the kille virus vaccine in 1955, changes in the epidemiology of poliomyelitis became apparen

Table 2

Reported Cases of Paralytic Poliomyelitis by Age Group, Race, and Immunization Status, North Carolina, 1959

White				
Age Group	Doses of Vaccine			
	0	1 or 2	3+	Total
0-4	53	18	11	82
5-14	16	9	14	39
15-44	29	7	1	37
Total	98	34	26	158

Non-White				
Age Group	Doses of Vaccine			
	0	1 or 2	3+	Total
0-4	49	16	4	69
5-14	26	6	6	38
15-44	10	2	0	12
Total	85	24	10	119

in North Carolina. The number of paralytic cases reported yearly dropped from 183 in 1955 to 37 in 1958. The age contributing the majority of the cases shifted from school age children to preschool children. The proportion of cases in the Negro rose from about 12 per cent in 1955 to 45 per cent in 1957. Age- and race- specific attack rates paralleled the changes in the number of cases. Data presented in table 1 show age- and race-specific attack rates for four age groups. All cases of poliomyelitis are included for 1940, 1948, and 1953; paralytic cases only are shown for 1955, 1957, and 1959.

Paralytic and nonparalytic cases

Of 314 cases of poliomyelitis reported during 1959, 277 were paralytic and 37 were nonparalytic. This was the largest number of paralytic cases reported since distinction between the two forms has been accurately reported. In table 2 the number of paralytic cases is shown by age group, race, and immunization status. More than one-third of these cases occurred among unimmunized children under the age of 5. This age group accounted for more than half the total number of reported paralytic cases. The age distribution is similar to that seen prior to 1944 and in 1957 and 1958.

Paralytic poliomyelitis was reported from 60 of 100 countries in the state in 1959. As indicated in figure 1, the preponderance of cases occurred in only a few counties. Tables 3 and 4 indicate the race-specific attack rates for paralytic poliomyelitis in some of these areas and indicate the type of virus associated with the outbreak.

Types of virus

More than a third of the cases reported in 1959 were confirmed by isolations of the virus. Type 1 virus was recovered from 88 cases in 35 counties. Type 3 virus, on the other hand, was recovered from 16 cases

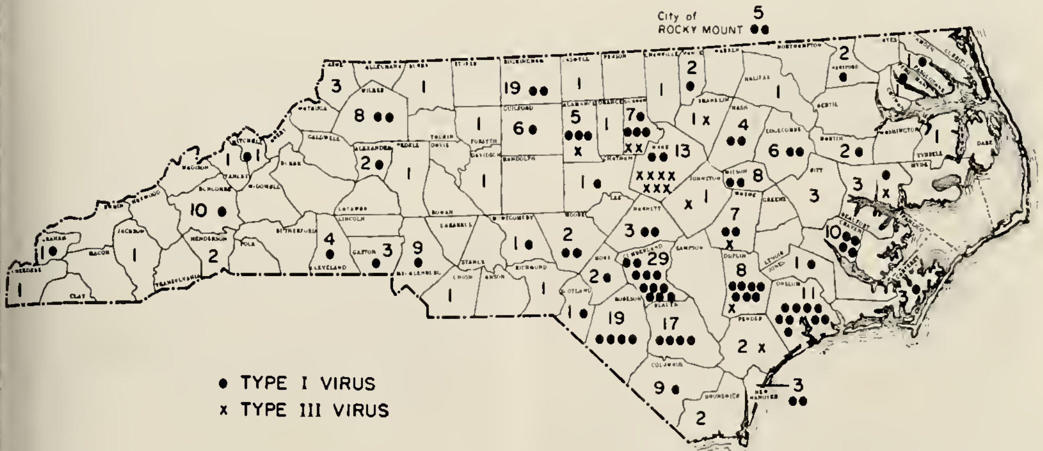

Fig. 1. Geographic distribution of cases of paralytic poliomyelitis and positive virus isolations in North Carolina, 1959.

Table 3

Race-Specific Attack Rates Per 100,000 Population and Predominant Virus Type For Paralytic Poliomyelitis in Selected Geographic Areas North Carolina, 1959

	Cases			Rates			
Area	W	NW	Total	W	NW	Total	Virus Type
Bladen County	2	15	17	10.6	113.5	54.4	1
Rockingham County	13	6	19	22.7	43.2	26.7	1
Wake County	4	9	13	3.4	20.9	8.1	3
Raleigh	2	4	6	3.2	20.1	7.2	3
Wayne County	5	2	7	12.2	6.9	10.0	1
Goldsboro	4		4	28.5		15.8	1
Wilson County	1	7	8	2.8	30.6	13.7	1
Wilson		4	4		36.8	15.1	1

Table 4

Cases and Race-Specific Attack Rates Per 100,000 Population and Predominant Virus Type of Paralytic Poliomyelitis for Geographic Areas Including Military Installations North Carolina, 1959

	Cases				Rates				
Area	W	NW	Military Dependents	Total	W	NW	Military Dependents	Total	Virus
Craven County	5	5	(4)	10	10.5	28.6		15.4	1
Cherry Point			(All cases reside off the Base proper)						
Cumberland County	21	8	(18)	29	21.6	24.4		22.3	1
Cumberland Co. less Ft. Bragg	16	7	(12)	23	16.5	21.3		17.7	
Fort Bragg	5	1	(6)	6			51.2	16.1	
Fayetteville	14	6	(10)	20	43.5	32.2		39.4	
Cumberland County less cases in Fayetteville & Fort Bragg	2	1	(2)	3	3.1	7.0		3.8	
Onslow County	11		(6)	11	19.6			17.1	1
Camp Lejeune	6		(6)	6			51.2	14.3	
Onslow Co. less Camp Lejeune	5		(3)	5	8.9			7.7	

Cases and Race-Specific Attack Rates Per 100,000 Population and Predominant Virus Type of Paralytic Poliomyelitis for Robeson County, North Carolina, 1959

Paralytic Cases				Rates per 100,000				
White	Negro	Indian	Total	White	Negro	Indian	Total	Virus Type
7	1	12	20	17.4	3.0	48.2	20.4	1

distributed in 9 counties. Type 2 virus was not recovered. The geographic distribution of virus types is shown in figure 1. Interestingly, type 3 virus was recovered from 7 paralytic cases in Wake County; but there seemed to be no epidemiologic correlation between cases, as they were widely scattered for the most part. Immunized individuals did not seem to account for an unexpectedly large proportion of cases attributable to type 3 virus.

Other viruses recovered included Coxsackie B-2, B-4, and B-5 and ECHO 1 and 9 Coxsackie B-4 virus was recovered repeatedly from the stool of a 10 year old gir who continued to have significant paralysis 60 days following the onset of illness This girl had received four doses of polio myelitis vaccine.

Studies of specific outbreaks

Several of the 1959 outbreaks were stud

ied in greater detail to determine if particular groups of individuals were contributing proportionately large numbers of cases, or if these outbreaks exhibited patterns different from those inferred from data for the entire state.

The Negro population of Bladen County provided the highest attack rate observed in the state. Investigation disclosed that most cases occurred in a rather small area of the county inhabited largely by rural Negroes. Contact between all but one of the cases in this county could be demonstrated. The association ranged from direct personal contact between cases to remote interfamily contact by a single member. In one instance the virus was apparently spread from one community to another by members of households in which there were no clinical cases.

Poliomyelitis outbreaks were observed in association with three of the four military installations in North Carolina. Data for the three counties in which these outbreaks occurred show that 31 of 50 cases were related to the military installations (table 4). Comparison of attack rates for Cumberland County, the site of Fort Bragg, showed that the vast majority of poliomyelitis cases in the county, other than those on the military reservation, were reported in the city of Fayetteville. Half of these occurred in military personnel or their dependents. In fact, 18 of 29 paralytic cases in this county occurred in persons associated with Fort Bragg.

The outbreak of paralytic poliomyelitis in Robeson County deserves special mention because the Indian population of this county contributed most of the cases.

Immunization status

Table 5 shows the immunization status of cases observed in these three outbreaks. Most cases occurred in unimmunized or in incompletely immunized individuals. The military dependents in Cumberland County might be considered relatively poorly immunized considering their ready access to vaccine.

The results of four poliomyelitis immunization surveys in North Carolina are shown in figures 2, 3 and 4. These surveys were conducted according to the quota sampling technique described by Serfling and others in 1959[4]. The percentage of individuals with three or more doses of killed virus vaccine and those with no vaccine is indicated for various age groups by race. The school age population, 5-14 years old, was by far the best immunized group in each area for both races. Variations in immunization status between areas for this group were quite small.

Table 5

Immunization Status of Cases of Poliomyelitis In Selected Population Groups And Geographic Areas

Population	Doses of Killed Virus Vaccine					
	0	1	2	3	4	Total
Cumberland County						
Military dependents	6†*	5	3†	4†		18
Civilian	5	2†	2	2		11
Robeson County						
Indian	8†		1	2		11
White	6			1		7
Bladen County						
Negro	11†††	2	2			15

* † Indicates one death

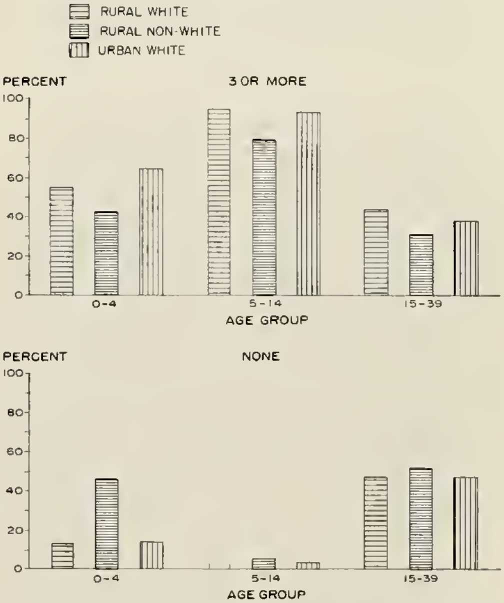

Fig. 2. Percentage of persons with three or more and those with no inoculations of killed virus vaccine, by age group, race, and area of residence—Halifax County, September, 1959.

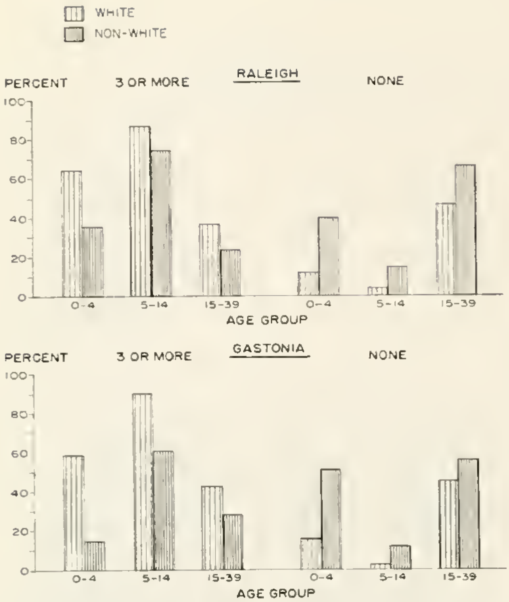

Fig. 3. Percentage of persons with three or more and those with no inoculations of killed virus vaccine by age group and race—Raleigh, January, 1960, and Gastonia, April, 1960.

Pre-school age Negroes were invariably the most poorly immunized group of children in each area surveyed. The group also provided the state's highest age-and race-specific attack rate.

Analysis of reports obtained 60 days or longer after onset of illness failed to reveal significant differences in residual paralysis between vaccinated and unvaccinated groups. Because of the small numbers in some of the categories, the status groups listed in the Surveillance Case Report were further combined for analysis. Complete recovery and minor involvement were grouped together, as were severe disability and death. Significant disability was treated independently. Patients in these three categories were analyzed according to those over and those under age 5, and according to those who had received no vaccine, those who had had one or two doses, and those who had had three or more doses. Of interest in this analysis is that 10 of the 21 deaths reported occurred in the adults 15 to 39 years of age. The case fatality rate for this group was 21 per cent.

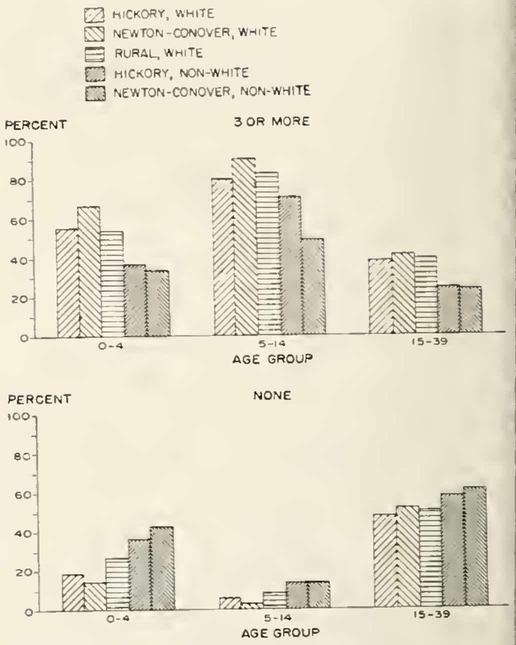

Fig. 4. Percentage of persons with three or more and those with no inoculations of killed virus vaccine by age group and race—Hickory, Newton-Conover, and rural Catawba County, June, 1960.

Estimate of vaccine effectiveness

Various estimates of the effectiveness of killed virus vaccine have been made. One is the use of effectiveness ratios calculated by determining the attack rates for specific age groups of unvaccinated persons, by applying these rates to the estimated vaccinated population to obtain an "expected" number of cases, and finally by comparing these expected numbers with the observed cases and calculating the percentage of reduction. It is believed that this method gives a less biased estimate of vaccine effectiveness than that obtained by calculating the relative reduction in expected cases from both the total population and the total number of observed cases. The former method has led to estimates ranging from 0.82 to 0.93 for three doses of killed virus vaccine, depending on the age group[5].

This estimation of effectiveness requires a valid estimate of the proportion of the population immunized. In North Carolina,

Table 6
Statewide Estimate of Immunization Status Against Poliomyelitis
By Age Group and Race, North Carolina, 1959

Age Group and Race		Doses of Killed Virus Vaccine			
		None	One or Two	Three or Moore	Unknown
0-4	White	17.2%	22.8%	59.5%	0.6%
	Non-white	39.5	23.4	35.1	1.9
5-14	White	4.1	8.3	87.4	0.1
	Non-white	9.2	16.7	71.4	2.7
5-39	White	48.1	10.5	39.7	1.7
	Non-white	56.6	11.2	27.3	4.7

Comparison of Attack Rate Ratio Non-White to White and Proportion of
Non-White to White Population with no Immunization by Age Group
North Carolina, 1959

Age Group and Race	Ratio, Non-White to White	
	Attack Rates	Unimmunized Population
0-4	2.0	2.3
5-14	2.4	2.2
15-44	1.0	1.2

he results of the four immunization sureys have been combined in table 6 to proide an estimate of the statewide immuniation status of the various age groups. Obections to this type of estimate are several. irst, equal numbers of individuals in each f several socioeconomic subgroups are seected for interview without regard for their native abundance," thus possibly weightng a survey in favor of highly different inority groups. Second, there is no assurnce that these areas are representative f the state as a whole, and are probably ot representative of the epidemic areas.

Because of the small variation between ocioeconomic subgroups in the proportion white children in the 5-to-14 age group ho have had three or more doses of killed irus vaccine, and because of the intensive hool immunization programs undertaken 1956 and 1957, this group was selected r the estimation of vaccine effectiveness. n this group, three or more doses of vacne have an effectiveness ratio of 0.96. This comparable with the figure 0.93, based on ational data, that has been proposed for is age group with three doses of vaccine, th figures being compatible with an efctiveness estimate of 80 to 90 per cent[5].

As a possible test of the validity of these sumptions, attack rate ratios between Negroes and whites were compared with the ratios between unimmunized Negroes and whites for these age groups. These are presented in table 6. There appears to be close correlation between the observed age- and race-specific attack rates and the estimated immunization status of these groups.

Conclusion

The examination of the 1959 poliomyelitis morbidity data in North Carolina showed some marked variations from previously observed patterns. There was a significant increase in the number of cases over that of the preceding four years. In the light of rather intensive immunization programs in most areas of the state, it would appear that lack of artificial immunity, while important in several areas of high incidence, was not the entire reason for this increased incidence. Type I poliovirus was found to be widespread through the state. Its association with the major epidemic areas and with sporadic cases indicates that virus prevalence played a significant role in the incidence of this disease.

It would appear that wide prevalence of virus, modified by the uneven pattern of artificial immunity, could well account for the 1959 poliomyelitis experience, as the new trends in age and race incidence appear to reflect deficiencies in the program of artificial immunization with killed virus vaccine.

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Cardiopulmonary Hypoxemia Encephalomalacia

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In the usual medical practice, hypoxemia is most commonly caused by cardiopulmonary disease. Acute cerebral hypoxia is a fairly frequent and sometimes fatal complication, but many types of cardiopulmonary dysfunction result in lesser degrees of hypoxemia and permit survival for prolonged periods. Relatively mild hypoxemia may then become chronic and produce multiple effects that can alter or obscure the underlying disease process. For example, when prolonged or repeated cerebral hypoxemia leads to encephalomalacia, the signs and symptoms of central nervous system disease may predominate in the clinical picture and overshadow those of cardiorespiratory disease. If the true situation is not recognized, this complication may easily be confused with other conditions. The following is a case in point.

Case Reports

Case 1

A 66 year old farmer had complained of "cigarette cough and chronic bronchitis" for many years. For several days during the first week of August, pleuritic pain in the right upper anterior part of the thorax was accompanied by increased cough with mucopurulent expectoration, generalized myalgia, anorexia, fever, and chills. Afternoon fever as high as 39.2° C. (102.5° F.) and night sweats persisted for the ensuing three weeks, as weight loss and dyspnea developed. On August 29 his attending physician obtained a chest roentgenogram which disclosed irregular, confluent, soft-appearing, nodula densities and cavities in the right upper lobe having the roentgen appearance of moderatel advanced, active pulmonary tuberculosis. Sputur examination, however, failed to establish th presence of tubercle bacilli until September when a consultation was obtained and a properl collected specimen demonstrated numerous aci fast bacilli with the morphologic characteristi **Mycobacterium tuberculosis.**

The patient was admitted to the Scott Parke Sanatorium on September 7, at which time e amination revealed an ill white male with blood pressure of 110 systolic, 70 diastolic, a pul rate of 96 per minute, and a temperature of 37. C. (100.0° F.). Tachypnea of 32 per minute was r corded, and he was judged to be in modera respiratory distress. The anteroposterior diame er of the thorax was moderately increased, a there was a slight kyphosis. Chest expansion a diaphragmatic excursions could not be accurate measured because of dyspnea and weakne Percussion resonance, intensity of breath soun and pectoriloquy were all slightly increased ov the right upper lung field, and variable coa and fine, moist rales were heard in the sa region. Cardiac enlargement was not detected percussion, but the neck veins were moderat distended in the semi-recumbent position, a there was tenderness over the liver, which w questionably palpable. No other relevant physi findings were noted. Admission diagnoses we (1) active, moderately advanced, pulmona tuberculosis; (2) chronic bronchitis with fibr and obstructive pulmonary emphysema; and cor pulmonale. The admitting physician rec nized that the patient was acutely ill, but parently did not judge him to be critically for no immediate treatment other than bed rest was prescribed.

By the following morning, however, there could be no doubt of the patient's critical condition, and the attending physician requested another medical consultation. At this time, the patient's breathing had become quite rapid and alternately shallow and gasping. The liver had become even more tender and definitely palpable, with a smooth, firm edge 6 cm. below the costal margin. Equally as alarming, he now exhibited motor asphasia and partial paralysis of the muscles of deglutition, resulting in choking and coughing whenever he attempted to swallow food or liquids. It was apparent that some neurologic complication had occurred, and foremost consideration was given to the possibilities of cerebral thrombosis, tuberculoma, or miliary spread with tuberculous meningitis. Since facilities were limited in this sanatorium for the care of such a patient, he was transferred to Duke Hospital by afternoon.

On admission to the hospital, the patient's temperature was 36.2 C. (91.1 F.) and his pulse rate 100 per minute. He was critically ill and obviously in respiratory distress, but physical examination contributed no new findings of significance. The question of cyanosis was raised but not resolved. The blood hemoglobin was 11.5 Gm., and the leukocyte count was 10,200 per cubic millimeter, with 80 per cent polymorphonuclear granulocytes, 8 per cent monocytes, 9 per cent large lymphocytes, and 3 per cent small lymphocytes. The Kahn, Kline, and Mazzini tests were negative. The patient died a few hours after transfer, the pulse rate rising to 120 per minute and the temperature to 37.5 C. (99.5 F.) terminally. Time did not allow the completion of other studies during life.

Autopsy findings

Postmortem examination disclosed the right side of the heart to be dilated and the liver congested. Both lungs were unusually marked by emphysema, being converted into series and clumps of large cysts and bullae interspersed with areas of normal-appearing alveolar tissue. Grossly, superimposed bronchopneumonia was extensively scattered throughout, and small, organizing thrombi or emboli were found in some of the smaller pulmonary arteries. A large area of characteristically caseous tuberculosis with a urious multilocular cavitation occupied virtually he entire two-thirds of the right upper lobe, the overlying pleura being greatly thickened by scar issue. Microscopically, the apical portion of the uberculous lesion consisted of fibrous tissue ontaining numerous hard tubercles and anthraotic pigment encircling an area of caseation, while lower in the lobe the alveoli were filled with caseating masses of monocytes and some polymorphonuclear neutrophils. The unusual multilocular cavitation was thought to have arisen from the rupture and aspiration of infected, caseous material into emphysematous bullae, resulting in massive necrotizing pneumonitis in a sensitized individual.

No tuberculosis was found outside of the lungs. The brain exhibited generalized and focal areas of edema, more marked in the right frontal lobe. There were multiple areas of acute and subacute focal necrosis of varying size and irregular shape distributed throughout the gray and white matter, more numerous in the cortical and thalamic regions. Microscopically, the older areas of necrosis were characterized by the presence of numerous macrophages, but there were no tuberculous lesions and no hemorrhages surrounding or infiltrating the necrotic sites. In view of the size, number, and focal distribution of these lesions, it was not believed that even multiple thrombi in small vessels could explain them. The cerebral changes were very similar to those seen in cases of delayed death after anoxia from high altitude or anesthesia accidents, and were interpreted as representing focal encephalomalacia due to prolonged hypoxemia secondary to profound pulmonary insufficiency.

Comment

Not only was this man's respiratory reserve greatly reduced by severe, chronic emphysema, but he also had fairly acute destruction of pulmonary tissue by a caseating tuberculous process with cavitation. Necrotizing bronchopneumonia contributed to pulmonary insufficiency, leading to pulmonary hypoxemia, edema and stasis, increased pulmonary vascular resistance, and cor pulmonale. The effects of this cardiopulmonary hypoxemia were even further compounded by an anemia, which, no doubt, contributed to the clinical uncertainty over the presence of cyanosis.

It is not surprising that this combination of factors resulted in cerebral hypoxemia and respiratory center dysfunction, leading to further cardiopulmonary and cerebral hypoxemia in an ever increasing interplay of forces. Had its development been more abrupt, it is likely that the patient would have died in an acute state of suffocation without neurologic complications. Yet, in spite of its eventual severity, hypoxemia developed relatively insidiously, and the patient survived long enough for encephalomalacia to develop. Because this complication was not anticipated, preventive measures were neglected, and because its manifestations were misinterpreted, its true nature was not perceived during life. Most clinicians have had similar cases.

Case 2

A 77 year old man was admitted to Duke Hospital with emphysema, decompensated hypertensive cardiovascular disease, and pulmonary edema. Response to digitalization and diuretics was slow, and a few days later he began to manifest mental confusion, nystagmus, transient tendon reflex changes, weakness on one side, and ataxic movements of the extremities. Eventually he died during a convulsion. Cerebral vascular embolization, thrombosis, or hemorrhage was clinically suspected, but autopsy disclosed focal areas of necrosis in the cortex, the pituitary, and the cerebellar and basal ganglia of the brain, which were attributed to hypoxemia encephalomalcia.

Case 3

A term delivery at Duke Hospital was complicated by 36 hours of labor terminating in a rotation and forceps extraction. After stimulants and oxygen, the infant's condition seemed satisfactory, but three hours later convulsive movements of the mouth and arms appeared. Shallow, grunting respiration was noted and weakness increased until death ensued several hours later. Traumatic brain damage was clinically suspected, but autopsy demonstrated congestive pulmonary edema and vacuolization of cytoplasm in many cells of the cerebral cortex. The pathologist attributed death to encephalomalacia from cardiopulmonary hypoxemia.

Discussion

Pathogenesis

Once it begins, hypoxemia tends to propagate itself because of two primary effects, each of which acts to intensify the other[1]. These two factors are respiratory center failure and cardiopulmonary dysfunction. First, normal impulse discharge governed by accumulation of carbon dioxide in the respiratory center leads to increased minute volume with a reduction in tidal volume and vital capacity, soon followed by irregular breathing and waxing and waning of tidal volume. Next, periodic deep breathing disappears, with loss of normal response to carbon dioxide stimulation, and the tidal volume is further reduced. Finally, rapid, shallow breathing supervenes, resulting in trifling, inefficient ventilation of the alveoli, which does little to move air into many lobules. Collateral ventilation through small air pores fails, and atelectasis of nonventilated air spaces occurs. This inefficient distribution of gases reduces the oxygen-diffusing capacity of the lung and produces a pattern of pulmonary dysfunction in the early phase similar to the syndrome of "alveolar-capillary block," in that the essential defect is an interference with the transfer of oxygen from the alveolus to arterial blood[2]. Evidence suggests that increasing anoxia soon leads to arteriolar constriction, increased pulmonary vascular resistance, pulmonary arterial hypertension, capillary stasis, and permeability of the endothelium to plasma water and solutes[3]. Fluid in the alveoli then leads to additional blockage of air, further local anoxia, and advancing pulmonary edema and dysfunction, as cerebral hypoxemia and respiratory center failure increase concomitantly.

Signs and symptoms

The signs of mild hypoxemia are usually not obvious unless sought, and when considered individually they are not specific for oxygen lack; but when they occur together or in sequence, they almost invariably become highly suggestive[4]. First and foremost is an increased respiratory rate and use of the accessory muscles of respiration. This may sometimes, but not necessarily always progress to the point of clinically apparen dyspnea. When this development is associated with an increased pulse rate, accompanied by an initial rise and fairly prompt secondary fall in blood pressure excessive sweating and pallor, the likelihoo of hypoxemia is very great. If restlessness apprehension, and clouding of the sensorium follow, the presence of some degre of hypoxemia is almost certain. Nevertheless, the entity must be suspected in orde to be recognized, for clinical manifestation of mild degrees of hypoxemia can be ver subtle, nonspecific, and confusing, particu arly in the common cardiopulmonary di eases.

The difficulty lies in the nonspecificity the symptoms and signs, for the patien with cardiopulmonary hypoxemia is ofte the very patient who already has some re piratory disturbance, in whom some circulatory or vascular abnormality might be expected, or in whom some evidence of senility or other neurologic complication might be likely anyway, so that the significance of the sequence of events can be easily discounted or misinterpreted. Cyanosis is an extremely untrustworthy guide, because of the many variables in different patients, observers, and their environments[5]. It is virtually worthless in the presence of anemia, since approximately 5.0 Gm. of reduced hemoglobin must be present in capillary blood to produce detectable cyanosis, and before this point is reached in an individual having less than the normal amount of total hemoglobin, the oxygen transport will already have become dangerously low.

Treatment

Definitive treatment is logically based upon the ultimate correction, insofar as possible, of some or all of the various underlying causes of hypoxia that may exist: (1) diminution of oxygen tension, as in high altitude or anesthesia accidents; (2) interference with oxygen uptake, as in airway obstruction, impaired ventilation, venoarterial shunts, or other abnormal cardiopulmonary mechanisms; (3) stagnation of blood, as in circulatory failure, cardiac arrhythmia, or vascular obstruction; (4) diminution of functioning hemoglobin, as in anemia; and (5) respiratory paralysis from toxic substances, as in alcohol, narcotic, or cyanide posioning.

Such a concept makes it obvious that if anemia is present; if too much sedation has been used; if cardiac arrhythmia or decompensation exists; if pulmonary infection, stasis, edema or atelectasis plays a part; if the atmosphere is vitiated by inert gases or lowered oxygen tension; or if hyperthyroidism or a veno-arterial shunt is suspected, attempts should be made as soon as possible to correct all—not just one—of these contributory factors as far as possible. In practice, of course, all these components are seldom completely correctable, but when prompt, appropriate action is taken, even partial or temporary improvement in one or more of them may turn the tide sufficiently to break the cycle of chronic cardiopulmonary hypoxemic propagation.

Energetically combatting one cause of hypoxia while overlooking the proper control of others is apt to invite failure, and by far the most common error in the management of mild or chronic hypoxemia is the failure to do more than prescribe oxygen. Unfortunately, reliance cannot be placed upon this measure alone, for by the time the need becomes clinically apparent, the cycle of hypoxemic propagation has already commenced and the administration of oxygen alone will not then insure adequate ventilatory effort, efficient respiration, and improved mixing of pulmonary gases. In fact, there is a special danger in the treatment of chronic hypoxemia, especially in emphysema with carbon dioxide retention, by the administration of high concentrations of oxygen, for this may cause further depression of a hypoxic, disfunctioning respiratory center which has lost its normal response to carbon dioxide accumulation[6]. Moreover, primarily owing to alterations in the mixing of alveolar air, even small changes in pulmonary ventilation will profoundly affect the arterial oxygen saturation, which may be raised from 10 to 20 per cent by increasing the resting ventilation by one-half[7]. This illustrates the fundamental importance of improving respiratory efficiency by clearing obstructed or impaired airways by medicinal, mechanical or surgical means, such as the bronchodilator, decongestant and mucolytic drugs, bronchoscopic aspiration, tracheotomy, postural drainage, coughing and expectorating mechanisms, intermittent positive pressure, breathing exercises, and even forced hyperventilation whenever applicable. With appropriate chemotherapy and other supportive media, such measures are safe and do not interfere with healing in pulmonary infections[8]. In spite of their logical indications and proven value, however, they are sometimes neglected or their use is delayed in clinical practice.

Even when it is obvious that pulmonary infection may interfere with respiratory efficiency, the pressing need for prompt and and thorough-going treatment is sometimes grossly understimated by the clinician unaware of the danger of hypoxemia. Not infrequently, also, the contribution made to hypoxemia by pulmonary or systemic circulatory abnormalities or by paralytic respiratory impairment is realized late[9]. A balanced perspective of thoroughness and effective timing in the application of corrective measures is required in the successful management of hypoxemia, and this is best achieved by a systematic and coordinated approach in terms of improving the delivery and mixing of oxygen in the alveoli, of increasing oxygen uptake and transport by the blood, and of improving respiratory center function.

The dangers of mild hypoxemia

The danger of death from severe anoxia is universally known, but because many persons recover every day from variously caused transient asphyxia without apparent after effects, mild hypoxemia is often not recognized as dangerous, and its cerebral effects are often regarded as temporary and reversible. Yet the importance and variety of neurologic complications encountered in chronic cardiopulmonary hypoxemia are demonstrated by the multiplicity and gravity of their clinical effects: impairment of consciousness and memory, confusion, euphoria, depression, slurring or scanning of speech, incoherence, somnolence, blindness, incontinence, hyperpyrexia, ataxia, flaccidity, spasticity, rigidity, reflex changes, and coma. In many postmortem reports, moreover, clinical manifestations of cerebral hypoxemia can be correlated with documented pathologic changes in the brain: vascular dilatation, cerebral edema, scattered minute hemorrhage or thrombi, ganglia degeneration, and focal or confluent areas of necrosis[10].

Owing to a difference in metabolic rates, different cells in the brain vary considerably in their susceptibility to oxygen lack, and animal experiments have shown that this susceptibility to anoxia increases with repetition or pronlongation of exposure to low oxygen tension[4]. Other experiments have demonstrated progressive and accelerated destruction of cerebral nerve cells in proportion to the aging of the animal as well as to the number and duration of exposures to mild anoxia, seeming to indicate that relatively slight hypoxemia has a cumulative effect on the brain[11]. In general, the cells in the supragranular laminae of the cortex have the highest metabolic rates and are the most vulnerable, being affected earliest by the slightest fall in oxygen tension, and usually being the most rapidly exhausted and destroyed.

The morphologic change in different nerve tissues, however, does not necessarily represent comparative differences in susceptibility of cells to oxygen lack, for vascular and chemical elements also play an important part in determining the severity, nature, and distribution of cellular damage, and these effects have a greater influence the longer the anoxemic process lasts[12]. Although more or less prolonged or recurrent hypoxemia is usually a prerequisite for pathologically detectable encephalomalacia, there is no doubt from experimental and clinical evidence that relatively mild hypoxemia can produce permanent brain damage. The degree of damage largely, though not entirely, depends upon the abruptness, severity and duration of anoxia, for the effects on the brain, as on other organs, are influenced also by the general physical state of the body.

In the clinical state, a combination of different types of mild anoxia seems to bring about a greater predilection for encephalomalacia than even more severe grades of any one type alone[4]. From a practical standpoint, if hypoxia from any cause persists for an appreciable time, it is almost always, regardless of the initial etiology, complicated in clinical practice by the development of a combination with cardiopulmonary hypoxemia; and a single, clear-cut type is the exception rather than the rule[1]. Thus, it is usual to find combinations of causes playing a role in hypoxemia, thereby increasing the risk of encephalomalacia. Susceptibility to encephalomalacia, therefore, should be especially anticipated in the chronic cardiopulmonary diseases, when emphysema, pulmonary infection, and circulatory failure often coexist, even though clear evidence of hypoxemia is lacking. It is at this stage that improvement of pulmonary, circulatory, or respiratory center efficiency can best be expected to help prevent permanent neurologic damage or death from hypoxemia encephalomalacia. Because the best opportunity for treatment is soon lost when the effects of hypoxemia become apparent, the prophylactic correction of all contributory components should be promptly and systematically planned and attempted, for the prevention of encephalomalacia is more practical and rewarding than is its treatment.

Summary

A case of encephalomalacia caused by chronic cardiopulmonary disease is presented, and attention is directed to the fact that such cases are not uncommon but are easily unrecognized. Some pertinent factors in the pathogenesis, recognition, and management of cardiopulmonary hypoxemia are discussed. The anticipation of encephalomalacia in chronic cardiopulmonary diseases and the prevention of its development by the logical management of hypoxemia is emphasized. The regrettable fact is that many cases of cardiopulmonary disease with various neurologic changes are probably wrongly diagnosed as irreversible cerebrovascular disease such as arteriosclerosis, thrombosis or hemorrhage, when the cerebral damage may actually be caused hy a process that is sometimes reversible or correctible—namely, chronic or repetitive, mild hypoxemia. Such hypoxemia is treacherous, and it deserves prompt, thorough treatment directed toward coordinated, systematic correction of all possible predisposing or related causes. It is possible that irreparable neurologic damage and death could be avoided in many cases by more widespread recognition of the danger of encephalomalacia from insidious hypoxemia in the common, chronic cardiopulmonary diseases.

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In worrying about our patients, there are a few simple principles to follow. When a diagnosis is definitely established and the course of the illness follows according to expectation, we can dismiss that problem from our mind. If a case of pneumonia responds promptly to antibiotic therapy and convalescence is favorable, we stop worrying about it. The same is true of incurable conditions that are obviously going downhill, such as metastatic carcinomatosis.—Levine, S. A.: Worry—Where Will It Get You? The Pharos 23: 213 (Oct.) 1960.

Postpartum Pituitary Necrosis

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Since 1914, when Simmonds described an autopsy performed on a woman with fibrosis of the pituitary gland, the medical profession has been aware of the term "pituitary cachexia" associated with pituitary destruction[1]. It was not until 1937, however, that Sheehan gave detailed descriptions of fresh infarcts in the anterior pituitary lobe of women dying from hemorrhage and collapse at childbirth. In the individuals so affected who survived, he related these lesions to the ultimate pituitary fibrosis reported by Simmonds and others. He further pointed out that cachexia is not necesarily a common finding in postpartum pituitary necrosis, and that up to 50 per cent of the anterior lobe can be destroyed without giving rise to symptoms. He believed that many of the milder cases of pituitary injury go undetected[3].

A suspected case of this milder type is reported here with the hope that it will focus attention on the problem, although the laboratory findings are inconclusive.

Case Report

The patient was a 23 year old white woman, gravida i, para i, whom we first saw in May, 1960, because of weight loss, amenorrhea, and chronic cough. The family history was negative except for the death of a grandfather from "cancer" of unkown origin. The system review and past medical history were negative until the patient became pregnant in October, 1958.

Past history

Her initial examination was not remarkable. The course of the pregnancy was uneventful until February 11, 1959, when she was admitted to Iredell Memorial Hospital with a diagnosis of "acute pyelitis, severe." Analysis of a catheterized urine specimen revealed a trace of albumin, a rare red blood cell, and many white cells. Treatment with tetracycline and sulfonamide drugs resulted in apparent clearing of the infection. The patient became afebrile and was discharged six days after admission. The hematocrit was then 33.

On February 22, 1959, the patient was readmitted with acute abdominal pain and iron deficiency anemia. The hematocrit was then 30. A surgical consultant believed that she had either appendicitis or acute pyelonephritis on the right. Operation was contemplated, but in view of the anemia it was thought best first to transfuse the patient with whole blood. After two transfusions without reaction the hematocrit was 35. By this time she had improved so much that she was discharged without operation.

Throughout the rest of the pregnancy she was given supplementary iron and vitamins. On July 1, 1959, she was readmitted and after an uneventful labor was delivered of a full-term male child. A left mediolateral episiotomy was done. There was never any evidence of clinical shock. Blood loss was estimated at 100 cc. The hematocrit was 36 on admission and 36.5 on discharge. She was given no ergot-type drugs while in the hospital. Testeed, one tablet twice daily, was given to supress lactation. She did well after delivery, and was discharged on the fourth day after admission.

Ten days later she was readmitted because of acute abdominal pain. She had become actutely ill the night before with a "shaking chill and high fever." On admission she was febrile and severely ill. All clinical findings indicated acute appendicitis. The hematocrit was 37.3 and the white blood cell count 18,000, with 96 per cent polymorphonuclears, 3 per cent lymphocytes, and 1 per cent monocytes. Operation was performed at once. The blood pressure remained stable at 120/70 throughout the operation. An acutely inflamed appendix covered by a purulent exudate, and small paraovarian cyst on the right were removed. The pathologist reported acute ulcerophlegmonous appendicitis and a paraovarian cyst. The postoperative course was uneventful, with no evidence of septicemia or serious complication and the patient was discharged after a hospital stay of 12 days.

She did well at home until September, 1959, when she failed to menstruate at the expected time; however she did not consult a physician. Amenorrhea persisted until May, 1960, when she was seen again by her obstetrician, who referred her to us.

Physical examination

Her weight had been 115 pounds **post partum** (maximum weight before pregnancy, 135 pounds). When we saw her she weighed only 88 pounds. Marked emaciation was associated with a waxy, yellow discoloration of the skin, giving her the appearance of an old woman. She stated that, although her appetite had not been robust, her dietary intake was not in keeping with her marked weight loss. A chronic nonproductive cough had been present for two or three months.

Tuberculosis or malignancy was suspected, and she was admitted to the hospital.

Despite the weight loss and cough, the lungs were clear to auscultation. The blood pressure was 90/60, and the radial pulse was regular at 70 per minute. Her temperature was 99.6 on admission, but thereafter tended to be subnormal.

Except for the extreme emaciation, the physical examination was not remarkable. There was slight, suggestive thinning of the pubic hair, but this finding was relative and difficult to evaluate accurately. Suspecting anorexia nervosa, we attempted to explore her past history and domestic life, but could find no unusual emotional or psychiatric difficulty. On the ward she ate well and seemed alert and cooperative at all times.

Accessory clinical findings

During the two days that she was in the hospital the following laboratory findings were recorded. A 12 lead electrocardiogram showed extremely low voltage complexes throughout, not in keeping with a thin young woman of 24. Since the conduction and configuration of the complexes otherwise appeared normal, this was thought to indicate possible hypothyroidism or severe malnutrition.

The basal metabolic rate was minus 15 per cent. The fasting blood sugar was 100 mg. per 100 ml., and the blood urea nitrogen 15 mg. per 100 ml. The hemoglobin was 13.4 Gm. and the hematocrit 43. The white blood cell count was 7,500, with 55 per cent neutrophils, 42 per cent lymphocytes, 3 per cent monocytes, and 2 per cent eosinophils. A VDRL test was negative. The sedimentation rate was 13 mm. per hour, corrected to 13 mm. The platelets appeared adequate. A specimen of voided urine showed a pH of 5.5, a specific gravity of 1.020, a trace of albumin, and 4 to 6 red blood cells and 8 to 10 white blood cells per high power field. The alkaline phosphatase was 3.25 units. The total bilirubin was 0.55 mg.

X-ray examination of the chest showed the bony thorax to be intact, and there were no demonstrable lesions or masses throughout either lung field. No destructive lesions were noted in the spine or pelvis. Examination of the skull in four projections showed the sella tursica to be within normal limits. No intracranial calcifications were noted. The pineal gland was not calcified. The skull was considered normal radiographically.

A 24-hour urinary assay for total neutral 17-ketosteroids was 4.2 mg. (normal range for a female, 6-15 mg.) The 17-hydroxycorticosteroid level was 4.4 mg. (normal for a female, 5-18 mg.). A protein-bound iodine determination obtained four weeks later was 4.2 micrograms, but was of no value because she was then on thyroid therapy.

It was our impression that the patient had pituitary necrosis with anterior pituitary hypofunction. There was no evidence of posterior pituitary failure or diabetes insipidus[4]. We also believed her to have adrenal cortex insufficiency and ovarian dysfunction, as indicated by the amenorrhea. Gonadotropins were not determined; however, in this syndrome the level of these hormones does not necessarily correlate with the presence or absence of amenorrhea[5]. Thyroid insufficiency was suspected but not definitely established.

Treatment and subsequent course

Substitution therapy consisting of 20 mg. of hydrocortisone once daily was started. After one week thyroid was added, ¼ grain daily increased gradually to ½ grain. On May 28 her weight was 93 pounds, and June 11 was 90¼ pounds. The hydrocortisone was then increased to 40 mg. daily. On July 20 there was noted improvement; her weight had increased to 96 pounds.

In July and August the patient noted some cramping at the expected time of menses. On September 17, 1960 she weighed 101½ pounds. At this time the hydrocortisone was stopped and she was given methylprednisolone, 4 mg. twice daily for two weeks, then reduced to 4 mg. once daily.

In October, November, and December she failed to keep her appointments, but reported by phone that she was doing well. In December she felt so well that she stopped all medication on her own volition.

In January, 1961, she returned to the office complaining of nausea and vomiting. She had taken no drugs since the first week in December. Her last menstrual period had been in November. Her weight was now 107 pounds and the blood pressure 112/70. With the 19-pound gain in weight, her appearance had improved greatly. Examination revealed softening of the cervix and uterine enlargement indicative of an early pregnancy. A frog test was positive. The blood sodium was 13.5 mg. and the blood potassium 5.9 mg. per 100 ml. The blood sugar, blood urea nitrogen, and other routine determinations were within normal limits. The protein-bound iodine was 4.8 micrograms. The urine assay at this time revealed the neutral 17-ketosteroids to be 8 mg. per 24 hours. The 17-corticosteroids were 3.9 mg. per 24 hours. The patient was placed on Tigan, 100 mg. three times daily before meals, for nausea, but no other drugs were prescribed.

Comment

Sheehan[6] has estimated that necrosis of 60 per cent of the anterior pituitary is required to produce slight symptoms and 75 per cent to produce moderate symptoms; 95 per cent usually produces severe symptoms. He also reported that a small pituitary scar was found at death in many women who had enjoyed good health following a delivery complicated by severe hemorrhage. At autopsy the necrosis and the resulting fibrosis are usually found to have spared the pars tuberalis, the part just in front of the attachment of the stalk, and a thin layer of small scattered islets just beneath the capsule. The posterior lobe is usually normal, but in clinically severe cases rarely as much as 10 per cent of the anterior lobe remains. Most of the remaining cells are chromophobes, although some eosinophils and occasional basophils may be present. The adrenal cortex is markedly atrophic, but the medulla often is not disturbed. The thyroid and gonads are generally atrophic, but the other ductless glands tend to be normal[2-8].

During the first few weeks after delivery the symptoms are not very striking. In severe cases there is no lactation, and the breasts involute rapidly. In the ensuing weeks manifestation of gonadal, thyroid, and adrenal insufficiency appear, often in that order. Although the menses cease, there is no menopausal reaction. Atrophy of the genitalia and loss of libido are common. Sensitivity to cold, loss of hair and teeth, brittleness of the nails, dry skin, drowsiness, loss of ambition, and constipation appear. Weakness, gastrointestinal disorders, and hypoglycemic reactions may occur. The patient may have mild symptoms for a number of years, then undergo rapid deterioration and death in a few months. In others the terminal phase is prolonged, sometimes for years, and the untreated patient becomes a permanent invalid. Some patients complain of weakness and fatigue; others maintain normal activity despite marked pituitary insufficiency.

The individual appears prematurely senile, with thin, smooth, elastic, cool, dry, pale, and slightly yellowish skin. The face may appear myxedematous, but can look normal. Hair on the head and body tends to be scanty. The axillary hair tends to decrease more rapidly than the pubic. The patient may or may not be cachectic. The temperature, blood pressure, and pulse rate tend to be subnormal. Glandular insufficiency may be demonstrated by a large number of tests.

Therapy consists of replacing the target gland deficiency, primarily the adrenal and thyroid. Care must be taken not to start the administration of thyroid prior to adequate adrenal replacement lest an Addisonian crisis be precipitated. Estimation of the follicle-stimulating hormone content of the urine is the test usually selected to exemplify the phenomenon. However, as Smith and Howard[5] pointed out, there may be not only a great variation in the degree of pituitary injury, but also a marked dissociation in the insufficiency of the thyroid, adrenal cortex, and gonads[7].

In our case, the cachexia was much more impressive than the laboratory data. Although there was no evidence of clinical shock, hemorrhage, or excessive use of ergot, there is no doubt that this patient had a severely septic appendix removed exactly 14 days *post partum*. This is a very critical time for possible pituitary injury. The first case of pituitary fibrosis, described by Simmonds in 1914, was in a woman who had a history of "puerperal sepsis."

Hypothyroidism was suggested but not proved in our patient. It is worth noting that thyroid activity can be normal in postpartum pituitary necrosis, and even hyperthyroidism has been reported in one well documented case[9]. The initial protein-bound iodine determination was of no value in our case, because thyroid replacement had already been started. The follicle-stimulating hormone assay of the urine was not available to us at the time we saw the patient, nor were we able to obtain a vaginal smear for determining the estrogenic effect. We were left with the clinical observation of amenorrhea occurring *post partum*, in association with the rest of the clinical picture and in the absence of any pelvic disease. As stated earlier, amenorrhea in this syndrome does not necessarily correlate with gonadotrophic levels. Although the adrenal hormones were definitely below normal, the deficiency was not great enough to be completely diagnostic. Stimulation with ACTH, accompanied by a slow, progressive rise of steroids, would have helped greatly in diagnosis.

Because the patient lived in a rural area area several miles away and hospitalizatio had to be held to a minimum, the amount of diagnostic study permitted was limited. In retrospect, I am sure we could have chosen our studies more wisely and efficiently. The fact that our patient is now pregnant indicates a spontaneous partial return of pituitary activity. It is hoped that the stimulus of pregnancy will further improve it and return her to a relatively normal state of health, as has been reported to occur[10].

Summary

We have reported what we believe to be a case of postpartum anterior pituitary necrosis following a septic complication (appendicitis) two weeks *post partum.* Although partial pituitary failure persisted for at least one year, improved function is indicated by the present pregnancy.

The patient exhibited marked cachexia despite the lack of really conclusive laboratory proof of pituitary failure. This lack is due in part to a poor choice of laboratory studies and inability to get the proper tests done at the proper time. Despite these obvious shortcomings, we believe that the case is sufficiently interesting to justify this report. Clinical awareness of this problem is certainly necessary if the milder and more atypical cases are to be adequately treated.

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If any of us were seriously sick, we would want our physician to worry about us. The man who attends his patient in his office or in the sick room, no matter how expert he may be, and then, dismisses the problem from his mind will overlook things from time to time. This need not matter if the problem proves to be trivial or self-limited. But when the situation is serious, not progressing favorably and not one from which a spontaneous recovery takes place, the physician must worry. This means that he should think about his unsolved problems when he is otherwise unoccupied, while driving his motor car or taking a stroll in the street. —Levine, S. A.: Worry—Where Will It Get You? The Pharos 23: 213 (Oct.) 1960.

The Senior Citizen - A Community Responsibility

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Considerable interest has been focused in the past few years, particularly and during the recent political campaign, on the subject of our aging population. The process of aging, which affects every living creature, begins the moment we are born and continues throughout our lives. During the process blood vessels will become arteriosclerotic, muscles will atrophy, and bones will grow osteoporotic; for aging is inevitable. But aging is a fact of life, and intelligent people accept its problems realistically. As interested physicians we should be concerned with how the problem has been thrust upon us, how long it will be with us, and what we can do about it. Recent events, in terms of the political climate of our country, have made it the concern of each of us.

Only within the last few decades has the aging population of our country become a pressing problem. The average life expectancy at the turn of the century was approximately 50 years. At present it is closer to 70 years. Today there are about 16 million persons in the United States who are 65 years of age or older. We are told that in 1970 the figure will be between 19 and 22 million. As our population increases, the proportion of older people will continue to rise.

These facts should be cause for rejoicing, not for alarm. However, we must temper our joy with realism. Having added years to millions of lives, our immediate concern should be to make those years as fully rewarding as possible. They should be satisfying years, and not years of grim endurance.

Needed—A Change in Attitude

Many factors have contributed to the extension of life expectancy: lowered infant mortality, better control of infections, reduced accident rates, improved nutrition, better living conditions, better surgery—the list could go on *ad infinitum*. From the standpoint of health, the aged have fewer problems than ever before. The physician, the dietitian, the bacteriologist, the biochemist—all have collaborated in the vast teamwork of medical research and contributed substantially to the reduction of disease. The senior citizen is the by-product of our healthier nation. The population explosion in this age group is what should vitally concern us as physicians. There has to be a change—a revolution, if you please—in the attitude toward our senior citizens.

To begin with, it must be accepted that the prolongation of life has not been achieved by medicine alone. It is the result of the efforts of many disciplines represented by industry, science, and government. Aging is a community responsibility—not an isolated medical problem. Our elderly people are living not only longer lives but healthier lives in these times. But—and this is a big BUT—good health is far more than the absence of disease and infirmity; it involves a positive state of physical, mental, and social well-being, and any factor that detracts from that well-being lessens the individual's total health.

We physicians can treat bacterial infections, remove diseased organs, and prevent certain diseases. But we will never be able to treat the elements of loneliness or rejection, or the lack of useful things to do. In this respect, all we can do is to diagnose the social maladies that produce the physical complaints of our patients. The real therapy must be administered by the patient's family, friends, church, and community—that complex group which we call "society". Society must be made to realize that aging in essence, is not a chronologic process, but a biologic psychologic and philosophic process. Aging varies with the individual and with his capacity to respond to his environemnt. The age old dilemma of Hamlet, "To be or not to be," should be the new attitude toward the senior citizens, not how many seconds, minutes, hours, days, years we can add to life. The object should be not to live as long as possible but to live as satisfyingly as possible, and, as a corollary, to withdraw from life as graciously as possible when the obvious facts require it.

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*Chairman, Committee on Chronic Illness, Mecklenburg County Medical Society.

The concept that aging is a time for rest and respite is misleading and contrary to biologic principles. The aged person has to continue to struggle, as is the case with any living cell. In the twilight years of life, however, this struggle should be invested with a vision of healthy maturity; one imbued with a positive attitude towards the problems of life. Chronologic age has little to do with a human being's capacity to contribute, though it is a handy piece of information to have available when one is planning birthday parties, voting for the first time, or applying for a driver's license. But except for its value as a vital statistic, chronologic age is relatively meaningless.

Unfortunately, however, our society today holds values that penalize the senior citizens and produce numerous problems. It is oriented toward youth rather than age. When a person attains a "success complex" in his family, in civic affairs, or in his profession during his youth and middle years and then is forced to retire at an arbitrary age, society de-emphasizes his importance within its own complex. This de-emphasis of the elderly person's importance in the structure of our society has led to a fundamental neglect of the immense resources that lie resident in our senior citizens. As active participants in our society, they can make contributions toward its betterment—contributions which, in many cases, lie resident in the central nervous system rather than the musculoskeletal system.

Our goal should be the attainment of a healthy maturity in the later years, with emphasis on worth-while and satisfying performance. Our aim should be to increase the function and the efficiency of our senior citizens, rather than just add to the number of minutes, hours, days, and years of their lives. They want to be self-reliant; to maintain their faith and pride in themselves. They want a helping hand, not a handout.

These 16 million people whom we talk about have only one thing in common: they all have attained the chronologic age of 65 years or older. Many are still employed, others retired; some find life satisfying, others find it empty and meaningless. Each of them is different, and should be treated as such.

Few who reach retirement age have lived useless lives. Most have contributed according to their individual capacities to their communities. Yet, as ridiculous as it sounds, countless Americans believe that 65 candles on the cake disqualifies a man for anything more than just "baby-sitting, whittling, or sleeping in the sun." This is shockingly evident in the field of employment, where the right to be useful is frequently abridged by a compulsory retirement policy. Firms that hire so carefully, taking into account an individual's training, skills, aptitudes, and potentialities, find nothing inconsistent in retiring a valuable employee by a chronologic rule of thumb. Retirement in itself, for those who wish it and are ready for it, is not what produces problems; it is arbitrary retirement, regardless of whether or not the employee is still capable of doing a job, that does the damage.

Although medical science has helped to prolong life for many people, it is the responsibility of society to see to it that these added years are a blessing instead of an empty reprieve. We must come to think in terms of the older person's many other needs, whether they be housing, recreation, or community understanding. Until we can do this we will be taking a piecemeal, hit-or-miss approach to a challenge that demands much effort. This why a sterotyped governmental approach, as represented in the current proposals to provide health care for the aged under federal controls, fails to reach the nucleus of the problem.

Two basic philosophies are represented in legislative approaches to this problem being made today. One is the welfare approach—represented by the Forand type of legislation. The other, represented by the Kerr-Mill Act, is by way of federal grants to states providing matching funds. One provides medical assistance to the aged on the basis of need; the other on the basis of right, regardless of need. The Forand approach would give those who are eligible for social security payments—most of whom are over 65—hospital, surgical, and nursing home care under government-controlled programs, regardless of private income or assets, and regardless of need.

The Kerr-Mills Act

Let us first direct our attention to the Kerr-Mills Act. This piece of congressional legislation was triggered initially by the introduction of the Forand bill in 1959, and was insured by the now established precedent of amending the Social Security Act every two years or more, specifically every election year. During the last two years a total of 103 bills designed to amend the Social Security Act have been introduced in Congress. Thirty-three of these dealt in some way with health care for the aged.

The Kerr-Mills bill was passed by Congress and signed by the President in September, 1960. It is primarily designed to provide medical assistance to the aged, and it accomplishes this in two ways: (1) by amending the Old Age Assistance program to provide an increase in federal matching funds for those states which have a medical payment plan and program known as Old Age Assistance; and (2) by establishing a new program of medical assistance for persons over 65 not presently receiving old age benefits, this to be known as the Medical Assistance Act. For purposes of distinguishing between the two programs, reference will be made to the Old Age Assistance program as OAA, and the new program as the MAA.

The major changes made by the Kerr-Mills Act with respect to the OAA program is the authorization of an increase in federal contributions to the states which have established vendor medical programs. The amount of increase a state can obtain is based on a rather complicated formula involving the state's relative per capita income and the average amount spent per OAA recipient. What we are particularly concerned with at this time is the new MAA program covering those older people who are not eligible for OAA. These are the people who have been implicated in the recent political debates—the so-called "medically indigent."

The only limit to MAA benefits is a minimal level. The bill can be as broad or as narrow as each state desires. In order to implement the federal Act, each state must submit to the Department of Health, Education and Welfare for approval a bill passed by the state legislature and applying uniformly throughout all counties of that state. In addition to certain broad qualifying provisions, the state must provide for the inclusion of some institutional and some non-institutional services. In this case, "institutional" means hospital payments and care, while "non-institutional" applies to nursing and boarding homes, home and outpatient care, diagnostic services, and the like. The law specifies a lengthy list of benefits that may be provided, including nearly all the services now available to persons under a private basis.

While there are two programs, the law specifically states that it cannot be construed to permit a state to have more than one plan for the aged. In other words, both programs would have to be administered by the same state agency—in North Carolina, the Department of Public Welfare. The matching formula under the MAA program is similar to that of OAA in that the percentage of federal contribution is based on the state's relative per capita income. However, MAA differs materially in that the only limitation of the federal contribution is the amount that the state is willing to appropriate for the program. In other words, the range of services, both institutional and non-institutional, is to be left up to each state, depending upon the services uniformly available and the amount appropriated by the state and county to match federal funds. The formula for matching funds is as follows: federal—77.46 per cent; state and county equally divided—22.45 per cent.

This is the broad base which the Kerr Mills Act provides for giving medical assistance to medically indigent aged citizens It is left entirely up the states to implemen the law within their own boundaries, and t include as much or as little of the provision as they deem fit, providing matching funds are made available.

The North Carolina State Medical Society recently submitted a bill to our own state Legislature which would provide for the implementation of the Kerr-Mills Act in this state. It would provide ample assistance to the medically indigent over 65, after suitable eligibility and means have been determined by responsible people. It covers both institutional and non-institutional care, as specified by the Act. At present the bill makes no provision for payment to doctors for professional services. This omission is entirely in accordance with the wishes of the doctors of our state, who only recently voted overwhelmingly to provide medical care for the medically indigent over 65 on a personal, doctor-to-patient basis, as has been done enthusiastically and cooperatively in the past.

The King Bill

Let us now consider the legislation proposed by the present Administration and presented to Congress as the King bill. This bill, like the Forand bill, calls for an increase in social security payroll taxes to finance the proposed benefits. The proposed increase of ½ per cent on all salaries up to $5,000 per year would yield about 1½ billion dollars per year when fully operated in 1963.

It is thought highly improbable that this rise in social security taxes will yield enough revenue to meet the cost of the proposed program. Moreover, social security taxes are already scheduled to reach 9 per cent of payroll by 1969 without the passage of the administration medical care bill and the proposed aid to education bill. If enacted, the King bill will result in tax payments by all citizens earning $5,000 a year of close to $500 per year by 1963, or roughly 10 per cent of payroll. These are tax burdens which the individual American is ill equipped to bear.

Let us turn our attention now to some of the benefits provided under the bill. The King bill principally provides for free medical services for persons over 65 who are eligible for social security or retirement payments, whether or not they are working, or whether or not a financial need exists. The bill provides for inpatient hospital services up to 90 days in excess of $10 for each of the first 90 days, and "full service course" for the remaining 81 days. It provides up to 180 days of nursing home care after hospitalization; this limited to 120 days if 90 days of hospital care have been received. The patient will be encouraged to make extensive use of nursing homes instead of the more expensive hospital facilities. The patient, however, can demand 180 days of nursing care after only one day of hospitalization, and abuse in this respect can easily be seen. It provides that all hospital outpatient diagnostic services costing over $20 be furnished by a doctor hired by the hospital rather than by one's personal physician. It provides for 240 daily visits of "Home Health Service."

Social Security tax structure

These are the provisions of the proposed bill. Now let us examine the social security tax structure which is to pay for them. Availability of full benefits to all presupposes a prosperous economy and full employment, with employers and employees contributing to the till. The bill presuppose the absence of recession or depression, during which the economy would suffer and unemployment would rise. This would necessarily reduce the funds available for payment of benefits.

Let us consider the Social Security System, which itself is in a financially precarious position. The following figures are taken from the statistical abstract of the United States in 1960. Despite the fact that social security taxes have risen, the Old Age and Survivors Insurance and Trust funds, which would be the financial mechanism used in the proposed program of health care for the aged, ran in the red from 1957 to 1959. During this three-year period the total assets of the fund dropped from 22 billion to 20 million at the end of 1959. The original architects of the Social Security Act invisioned a maximum social security tax of 6 per cent, this on a tax base of $3,000 per year. In 1960 the social se-

Concerning Your Health and Your Income

THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA
SPECIAL GROUP ACCIDENT AND HEALTH PLAN
IN EFFECT SINCE 1940

This is our 21st year of service to the Society. It is our aim to continue to lead the field in prov ing Society members with disability protection and claim services as modern as tomorrow.

SPECIAL FEATURES ARE:

1. Up to a possible 7 years for each sickness (no confinement required).
2. Pays up to Lifetime for accident.
3. New Maximum limit of $650.00 per month income while disabled.

All **new** applicants, and those now insured, who are under 55, and in good health, are eligible apply for the new and extensive protection against sickness and accident.

BENEFITS AND RATES AVAILABLE UNDER NEW PLAN

Accidental Death Coverage	*Dismemberment Loss of Sight, Speech or Hearing	Accident and Sickness Benefits	COST UNTIL AGE 35 Annual Premium	COST UNTIL AGE 35 Semi-Annual Premium	COST FOR AGES 35 T Annual Premium	COST FOR AGES 35 T Semi-An Premi
5,000	5,000 to 10,000	50.00 Weekly	$ 78.00	$ 39.50	$104.00	$ 52.
5,000	7,500 to 15,000	75.00 Weekly	114.00	57.50	152.00	76.
5,000	10,000 to 20,000	100.00 Weekly	150.00	75.50	200.00	100.
5,000	12,500 to 25,000	125.00 Weekly	186.00	93.50	248.00	124.
5,000	15,000 to 30,000	150.00 Weekly	222.00	111.50	296.00	148.

*Amount payable depends upon the nature of the loss as set forth in the policy.

OPTIONAL HOSPITAL COVERAGE: Members under age 60 in good health may apply for $2 daily hospital benefit—Premium $20.00 semi-annually or $40.00 annually.

Write, or call us collect (Durham 682-5497) for assistance
or information

ALL CLAIMS ARE PAID IMMEDIATELY FROM OUR OFFICE.

Administered by
J. L. CRUMPTON, State Mgr.
Professional Group Disability Division
Box 147, Durham, N. C.
J. Slade Crumpton, Field Representative

UNDERWRITTEN BY THE COMMERCIAL INSURANCE COMPANY OF NEWARK, N. J.
Originator and pioneer in professional group disability plans.

AN IMPORTANT STATEMENT

TO MEMBERS OF THE MEDICAL SOCIETY

HOSPITAL SAVING ASSOCIATION

Your Blue Shield Plan

values the confidence and support of the physicians of North Carolina. Hospital Saving Association has cooperated fully with the Medical Society through a mutual Statement of Understanding entered into several years ago by both parties.

- Since it was nationally approved in 1946, the Association has conformed to all national requirements and cooperated with other Blue Shield Plans throughout the nation.
- The official Blue Shield emblem symbolizes national approval. For the Association, it also represents a cherished asset, one that has been built through untiring efforts, promotional expenditures—and with the understanding and cooperation of the Society itself for the past fifteen years.
- Through its extensive statewide sales organization, the Association continues to make Blue Shield available to North Carolinians. It is proud of its 1960 increase of over 61,000 participants—a gain of 12.78% over 1959.

In 1940, at the request of Dr. I. H. Manning, the House of Delegates permitted Hospital Saving Association to write Surgical and Medical programs. Thus, from the beginning, the Association has operated under the aegis of Medical Society leadership.

In 1946, Hospital Saving was included in the first group of Plans that were given the privilege of using the copyrighted Blue Shield emblem. National approval has been continuous; Hospital Saving has always considered it a sacred trust to protect and build the fine national and local concepts of Blue Shield.

More recently, the Medical Society instructed the Association to provide the DOCTORS PROGRAM of service benefits, which has grown in public acceptance to the point where over 31% of the Association's membership—and over 36% of its professional claims—are now under this program.

curity tax did go up as planned to 6 per cent, and the tax base already up to $4,800 per year. Under existing plans the tax is already scheduled to reach 9 per cent of payroll by 1969, and despite these increases, it is estimated that the new rise will not yield enough revenue to meet the cost of a medical care program to cover some 14 to 16 million people. Millions of young workers, who generally need every dollar to meet present obligations, would be taxed for the rest of their working days to pay for free medical care for the aged, including those willing and able to take care of themselves.

Why Doctors Support the Kerr-Mills Act

Why should interested physicians encourage the implementation of the Kerr-Mills Act rather than the King type of legislation? Because this law provides assistance to those persons 65 years of age or over who need it, and not as a benefit by right as proposed by the King bill. The need of medical assistance for the aged has been grossly exaggerated. Our State Medical Society has been trying to amass figures relative to the exact need of the elderly population in this state. Certain governmental agencies have estimated that some 140,000 North Carolinians over the age of 65 can be considered medically indigent. The State Society has figures to show that a more accurate estimate would be about 68,000 people. These figures have been difficult to obtain, but all concerned believe them to be accurate.

The total state population of people over 65 years of age is some 314,000 as of January, 1961. It is estimated that 12 per cent of them are certified beneficiaries of OAA. It is also estimated that a total of 188,000 can and would buy their own medical care, while only some 68,000 would qualify as medically indigent. In addition, it has been estimated that of these 68,000, approximately 8,000 would require hospitalization during any one year. These are the statistics that should concern us rather than the grossly exaggerated figures that have been distributed. These 8,000 are the people who need our assistance, and these are the people who can best be aided by a legislative vehicle such as the Kerr-Mills Act.

Let us analyze two bills in terms of cost. The cost of the King bill has been estimated to be close to 3.6 billion per year for hospitalization benefits alone. The Kerr-Mills bill has been estimated at a cost of 2 billion per year for all expanded health services, or four times less than the King bill would cost if all services were included.

Let us compare them in terms of quality of care. The King bill, it is felt, would lower the standards of medical care and services by massive use and abuse of existing facilities and personnel. The Kerr-Mills bill should preserve and maintain the present high standards of medical services, as it would be controlled somewhat by local government.

Let us compare the bills in terms of political philosophy. The King bill proposes to place the practice of medicine under the control of the federal government by the intercession of a third party in the patient-physician relationship. The Kerr-Mills Act encourages self-reliance and discipline in the private practice of medicine, and largely preserves the doctor-patient relationship while providing help for all who are in actual need of medical aid.

Let us compare the services offered. The Kerr-Mills Act allows each state to set up a full range of services depending upon state and local appropriations to match federal funds. It makes possible a shift from one source to the other for the sake of efficiency and economy as the program develops with experience. This comparison, we believe, eloquently demonstrates the relative values of both philosophies.

Now why should we, as interested physicians, oppose the King approach? We should oppose it because it is totally unnecessary: The Kerr-Mills Act, which is the law of the land, can do the job; the King type of legislation would result in poorer, not better health care for the aged, with government employees telling physicians what treatment they could provide for their patients, telling hospitals how to operate, telling nursing homes what they could and could not do. It would lead to the decline, if not the end, of private health insurance; it would inevitably expand into a compulsory national health service for every American; it would be staggeringly expensive. Most important, it is well to remember that such legislative vehicles as represented by the King bill are irreversible in nature and hard to stop. The tendency is to expand and never to contract them; and as time went on Congress would face continual pressure for more extended coverage and greater benefits, and costs would rise proportionately.

As a result of this thinking, the North Carolina State Medical Society recently voted overwhelmingly to implement the Kerr-Mills Act and to support legislative action to implement it in our state. Seldom has a program such as the Kerr-Mills bill offers been tailored so exactly to meet the specifications of providing medical assistance for those who need it rather than as a right for all.

Conclusion

Now where does the answer to the problem of providing the best possible health care for the elderly lie?

It lies in community action. It lies in the cooperative efforts of private citizens, health professions, and all parameter disciplines to provide for our senior citizens, not only medical care but retirement villages, new nursing homes, chronic disease centers, home care programs, recreation facilities, and research programs. In addition, the problem of financing the health care of our elderly is being met by private insurance and prepayment plans. Tremendous advances are being made in the field of health insurance. According to the Health Insurance Institute of America, 60 per cent of our senior citizens who want and need health insurance will have protection by the end of this year, and the percentages will increase until 90 per cent are covered by 1970. Moreover, particular hospital and medical needs of the aged are being met through the voluntary efforts of private citizens at the community level.

Much is being done, but much remains to be done. It is through the efforts of indiivdual communities as we apply ourselves to the mutual effort—which is considerable—that the day will come when every senior citizen in the United States will have the opportunity to find the richness and meaning, both in health and community understanding, that should make these twilight years the most rewarding years of his life.

SPECIAL REPORT

Report on Action of the House of Delegates
American Medical Association
110th Annual Meeting
June 25-30, 1961
New York City

Osteopathy, medical discipline, communications, surgical assistants, drug legislation, general practice residencies, relations with allied health professions and services, and poliomyelitis vaccine were among the major subjects covered by 115 resolutions and 28 reports acted upon by the House of Delegate at the American Medical Association's One Hundred Tenth Annual Meeting held June 25-30 in New York City.

Dr. George M. Fister of Ogden, Utah, member of the A.M.A. Board of Trustees and previously a member of the House of Delegates, was named president-elect of the Association. Dr. Fister will become president at the June, 1962, annual meeting in Chicago, succeeding Dr. Leonard W. Larson of Bismarck, North Dakota, who assumed office at the Tuesday night inaugural ceremony in New York.

The A.M.A. 1961 Distinguished Service Award was voted to Dr. Walter H. Judd of Minneapolis, physician and member of Congress, for his contributions as a medical missionary, humanitarian, and statesman devoted to world peace.

Total registration through Thursday, with half a day of the meeting still remaining, had reached 56,315, including 22,681 physicians.

This report was forwarded to the JOURNAL by Dr. Elias Faison, Secretary of the North Carolina delegates to the A.M.A.

Osteopathy

In considering a report of the Judical Council and three resolutions on the subject of osteopathy, the House of Delegates agreed with the intent of the report and resolutions, but instead adopted the following statement of A.M.A. policy:

"1. There can never be an ethical relationship between a doctor of medicine and a cultist, that is, one who does not practice a system of healing founded on a scientific basis.

"2. There can never be a majority party and a minority party in any science. There cannot be two distinct sciences of medicine or two different, yet equally valid systems of medical practice.

"3. Recognition should be given to the transition presently occurring in osteopathy, which is evidence of an attempt by a significant number of those practicing osteopathic medicine to give their patients scientific medical care. This transition should be encouraged so that the evolutionary process can be expedited.

"4. It is appropriate for the American Medical Association to *reappraise its application of policy* regarding relationships with doctors of osteopathy, in view of the transition of osteopathy into osteopathic medicine, in view of the fact that the colleges of osteopathy have modeled their curricula after medical schools, in view of the almost complete lack of osteopathic literature and the reliance of osteopaths on and use of medical literature, and in view of the fact that many doctors of osteopathy are no longer practicing osteopathy.

"5. Policy should now be applied individually at state level according to the facts as they exist. Heretofore, this policy has been applied collectively at national level. The test now should be: Does the individual doctor of osteopathy practice osteopathy, or does he in fact practice a method of healing founded on a scientific basis? If he practices osteopathy, he practices a cult system of healing and all voluntary professional associations with him are unethical. If he bases his practice on the same scientific principles as those adhered to by members of the American Medical Association, voluntary professional relationships with him should not be deemed unethical."

Medical Discipline

In a major move designed to strengthen the profession's disciplinary mechanisms, the House approved the conclusions and recommendations of the Medical Disciplinary Committee, with only three word changes. The House discharged the committee with thanks and commendation and directed that its functions be assumed as a continuing activity of the Judicial Council.

One recommendation suggests that "The bylaws of the American Medical Association be changed to confer original jurisdiction on the Association to suspend or revoke the A.M.A. membership of a physician guilty of a violation of the Principles of Medical Ethics or the ethical policy of the American Medical Association regardless of whether action has been taken against him at local level."

Another "encourages and urges that each state association report annually to the American Medical Association all major disciplinary actions taken within its jurisdiction during the preceding calendar year."

The report urged state and county medical societies to utilize grievance committees as "grand juries" to initiate action against an offender so as to obviate the necessity of making an individual member of a medical society complain against a fellow member.

The House suggested that each medical school develop and present a required course in ethics and socio-economic principles, and that each state board of medical examiners include questions on ethics and proper socio-economic practices in all examinations for license.

The report concluded with a recommendation that "American medicine at the national, state and local maintain an active, aggressive and continuing interest in medical disciplinary matters so that, by a demonstration of good faith, medicine will be permitted to continue to discipline its own members when necessary."

Communications

Acting upon four resolutions related to the Association's public relations program, the House adopted a substitute resolution directing the Speaker of the House of Delegates to name seven elected members of the House as a special committee "to study and continually advise the Board of Trustees on the broad planning and coordination of all phases of communications of the American Medical Association, so that the public and the members of the medical profession are properly and adequately advised of the policies and concern of the medical profession with respect to all phases and aspects of medical care for all people."

The House agreed with a reference committee opinion that "we have a very adequate Division within the A.M.A. capable of implementing any program of communications." The approved committee report also said that "the Communications Division of the A.M.A. needs the active support and cooperation of the House and of all members of the Association."

Surgical Assistants

In considering a Board report and two resolutions on the subject of surgical assistant's fees, the House approved the following five basic pricinples developed by the Judicial Council and the Council on Medical Service:

"1. Each member of the A.M.A. is expected to observe the Principles of Medical Ethics in every aspect of his professional practice.

"2. Each doctor engaged in the care of the patient is entitled to compensation commensurate with the value of the services he has personally rendered.

"3. No doctor should bill or be paid for a service which he does not perform; mere referral does not constitute a professional service for which a professional charge should be made or for which a fee may be ethically paid or received.

"4. It is ethically permissible for a surgeon to employ other physicians to assist him in the performance of a surgical procedure and to pay a reasonable amount for such assistance.

"This principle applies whether or not an assisting physician is the referring doctor and whether he is on a per-case or full-time basis. The controlling factor is the status of the assisting physician. If the practice is a subterfuge to split fees or to divide an insurance benefit, or if the physician is not actually employed and used as a bona fide assistant, then the practice is contrary to ethical principles.

"5. Under all other circumstances where services are rendered by more than one physician, each physician should submit his own bill to the patient and be compensated separately."

Efficacy of Drugs

The House strongly endorsed a Board report which pointed out the problems that would result from amending the Food, Drug and Cosmetic Act to authorize the Food and Drug Administration to determine the efficacy, as well as the safety, of a prescription drug prior to the approval of a new application. The A.M.A. will oppose such legislation before the Kefauver Committee, the report pointed out, on the basis that "a decision with respect to the effectiveness of drugs is dependent upon extended research, experimentation and usage." The House agreed that vesting such authority in the Food and Drug Administration would operate to limit research, the marketing of drugs and the exercise of discretion by the medical profession. "The marketing of a relatively useless drug is infinitely less serious than would be the arbitary exclusion from the market of a drug that might have been life saving for many persons," the House declared.

General Practice Residencies

Eight resolutions were introduced on the subject of creating new two-story, residency training programs in general practice. The House agreed that there appears to be a need for such programs for those individuals who desire more experience in obstetrics and surgery than may be available in the currently existing Family Practice Program. It approved a substitute resolution directing the Council on Medical Education and Hospitals to consider for approval other two-year programs in general practice which incorporate experience in obstetrics and surgery. The Council will review these programs on the basis of their individual merits and conduct a long-range evaluation of the new programs as well as the previously established Family Practice Programs.

Relations With Other Health Professions and Services

The House considered a Board report and 12 resolutions dealing with various aspects of medicine's relationships with allied health professions and services, including optometry. The Board report recommended the creation of a new A.M.A. Council to handle all the problems involved. The House, however, accepted a reference committee suggestion for establishment of a new Commission to Coordinate the Relationships of Medicine with Allied Health Professions and Services. The Commission will be composed of seven members appointed by the Speaker of the House. Subcommittees, composed of from three to five members selected by the Commission from lists of names submitted by the scientific sections, will consider problems in specific areas. The Commission will correlate and catalogue the reports of the subcommittees and will act as liaison agent between the subcommittees and those A.M.A. Councils where there may be overlapping interests.

Polio Vaccine

The House approved a report by the Council on Drugs on the present status of poliomyelitis vaccination in the United States and urged that it be made available to all physicians through the most effective communications media. The report clearly outlines procedures recommended for implementation of mass vaccination with the new oral vaccine when it becomes available. The House complimented the Council on its "clear and succinct statement on the initiation of the new campaign which will be needed to promote the new vaccine." The House agreed that the report provides the practicing physician with a reliable series of answers to the many questions which will arise during the change-over from Salk vaccine to oral vaccine. The report emphasizes, however, that "physicians should encourage, support and extend the use of Salk vaccine on the widest possible scale at least until the oral polio-virus vaccines currently under development and clinical trial become available."

Miscellaneous Actions

In dealing with resolutions and reports on a wide variety of other subjects, the House also:

Approved the "*Guides to Physician Relationships with Medical Care Plans,*" submitted by the Council on Medical Service, with these two changes: deletion of item 5 under "Responsibilities of the Medical Society," which said "To recognize that properly qualified physicians employed by, or otherwise serving, medical care plans should not be denied professional rights and privileges because of their service to such plans," and addition of a new item 1 under "Responsibilities of the Medical Care Plan," which reads:: "To provide the beneficiary of the plan with free choice of qualified physicians";

Reaffirmed its support of the *Kerr-Mills* program for the needy and near-needy aged and its opposition to any legislation of the *King-Anderson* type, declaring that the medical profession "will not be a willing party to implementing any system which we believe to be detrimental to the public welfare";

Approved a markedly expanded *drug information program* submitted by the Board of Trustees and the Council on Drugs;

Adopted the final report of the *Special Study Committee* of the Council on Medical Education and Hospitals and recommended that copies be sent to all medical school deans in the United States;

Decided to hold the *1963 Clinical Meeting* in Portland, Oregon, instead of Las Vegas, Nevada, as recommended by the Board;

Approved a plan by the A.M.A. Department of International Health to cooperate in the recruitment of volunteer physicians for emergency medical service in *foreign mission fields;*

Agreed to an increase of $20 in the annual A.M.A. *membership dues* to be implemented over a period of two years: $10 on January 1, 1962, and $10 additional on January 1, 1963;

Discontinued the Association's *General Practitioner of the Year* award;

Opposed legislative and administrative mandates which would compel physicians to prescribe drugs, or require pharmaceuticals to be sold, by *generic names* only;

Reaffirmed the Association's opposition to compulsory inclusion of physicians under the *Social Security* system;

Urged immediate legislation that will provide strong economic motivation for the construction and maintenance of *fallout shelters;*

Disapproved two resolutions which would have discontinued the scientific activities at the *Clinical Meeting*:

Urged *immunization campaigns* against both tetanus and influenza, and

Asked state and county medical societies to give full support to the *First National Congress on Medical Quackery* to be jointly sponsored next October 6-7 in Washington, D. C., by the A.M.A. and the Food and Drug Administration.

Open Session

At the opening session on Monday, Dr. E. Vincent Askey of Los Angeles, retiring A.M.A. president, challenged physicians and medical organizations to re-examine their own efforts to strengthen and improve medicine, and he warned against defeatism and failure to accept personal responsibility for answering criticisms. Dr. Larson, then president-elect, called on the profession to strengthen methods of self-discipline in both the state and county societies, adding that physicians must be concerned with improper or incompetent practice and unethical actions of all kinds. The 1961 Goldberger Award in Clinical Nutrition was presented to Dr. Frederick J. Stare, chairman of the Department of Nutrition at Harvard Medical School.

Inaugural Ceremony

Dr. Larson, in his inaugural address Tuesday night, said that the really good doctor, guided by the profession spirit, will always remember that medicine exists for just one purpose—to serve humanity. When the essence of that spirit is diluted or destroyed, either in a individual physician or in a nation, he added, medicine ceases to be a profession in the highest sense of the word. Dr. Larson also presented the Distinguished Service Award medal to Rep. Judd. Entertainment highlight of the inaugural program was a concert by the Montgomery County Medical Society Glee Club of Dayton, Ohio.

Election of Officers

In addition to Dr. Fister, the new president-elect, the following officers were named at the Thursday session:

Dr. Eustace A. Allen of Atlanta, vice president; Dr. Norman A. Welch of Boston, re-elected speaker of the House, and Dr. Milford O. Rouse of Dallas, Texas, re-elected vice speaker.

Elected to the Board of Trustees were Dr. Wesley W. Hall of Reno, Nevada, to succeed Dr. Fister; Dr. Homer L. Pearson, Jr., of Miami, Florida, to replace Dr. Julian P. Price of Florence, South Carolina, and Dr. Charles L. Hudson of Cleveland, Ohio, to fill out the term of the late Dr. Cleon A. Nafe of Indianapolis. The Board named the following officers: chairman, Dr. Hugh Hussey of Washington, D. C.; vice chairman, Dr. Percy Hopkins of Chicago, and secretary, Dr. James Z. Appel of Lancaster, Pennsylvania.

Named to the Judicial Council were Dr. Robertson Ward of San Francisco, to succeed himself, and Dr. Elmer G. Shelley of North East, Pennsylvania, to replace Dr. Pearson.

Re-elected to the Council on Constitution and Bylaws was Dr. Walter E. Vest of Huntington, West Virginia.

New Members of the Council on Medical Service are Dr. Charles Ashworth of Providence, Rhode Island, succeeding Dr. Carlton Wertz of Buffalo, New York, and Dr.

Burtis E. Montgomery of Harrisburg, Illinois, to succeed Dr. Charles Hudson of Cleveland.

For the Council on Medical Education and Hospitals, Dr. Dwight L. Wilbur of San Francisco was elected to succeed Dr. John W. Cline of the same city, and Dr. Kenneth C. Sawyer of Denver, Colorado, was named to succeed Dr. Guy A. Caldwell of New Orleans.

F. J. L. Blasingame, M.D.
Executive Vice President
American Medical Association

Report from The Duke University Poison Control Center

Jay M. Arena, M.D.
Director

Salicylate

Aspirin, like certain other drugs, has been deliciously flavored to make it acceptable to children, but as a result they readily mistake it for candy. Methyl salicylate (oil of wintergreen) is so highly attractive in appearance that children have been enticed into drinking it. In addition to these causes of salicylate poisoning, parents are sometimes guilty of overtreatment. They know that aspirin is beneficial in a febrile illness, but most of them are not aware that there is a rule of safe dosage: For each year up to the age of 5, children may be given 65 mg. (1 grain) every four hours—not more.

Continued administration of salicylates can lead to cumulative toxicity. Administration to an infant who is dehydrated, who takes little fluid, or who has reduced urinary output calls for the skill and knowledge of a physician.

The salicylates (methyl salicylate excepted) are rapidly and well absorbed from the digestive tract, including the stomach, and are found in all tissues afterward. Elimination is largely via the kidney, starting as early as 10 to 15 minutes after a therapeutic dose and continuing for several hours, but traces can appear in the urine for three days or more. About 20 per cent of the salicylate is oxidized in the tissues, and 70 per cent excreted in the urine.

Methyl salicylate has other characteristics which are of practical importance. It contains more salicylate at a time than other remedies: one teaspoonful (5 ml.) is equivalent to about 12 aspirin tablets (325 mg. each). The absorption of methyl salicylate may be delayed for hours; consequently, when oil of wintergreen has been swallowed, gastric lavage is almost always worth the trouble, even hours later.

Pharmacologic Effects

The pharmacologic effects of salicylates in large doses are fairly well, though not completely, understood. They consist in the main of initial hyperventilation, followed in severe cases by a metabolic acidosis. The first sign noted is rapid, deep and pauseless breathing. This hyperventilation is ascribed to some direct stimulatory effect on the respiratory center, and its result is excessive removal of carbon dioxide. With the loss of carbon dioxide there is primary reduction of the serum CO_2 level and a rise in the blood pH. That is, there is first a respiratory alkalosis, alleviated to a measurable degree by a compensatory renal retention of chlorides and excretion of sodium. In adults and older children the matter ends there, and (if the drug is withdrawn) recovery ensues within a few days.

In infants, and in others receiving disproportionately large doses, a metabolic acidosis next develops, and the pH falls to normal and then below. Among the factors producing the acidosis may be included some loss of sodium via the urine during the initial alkalosis, and, to some extent, the ketosis of dehydration, starvation, and fever; but these (and the accumulation of salicylate ion) are not regarded as the entire explanation. It has been suggested that an effect of salicylate may be a metabolic change at the cellular level, so that organic acid metabolites accumulate apart from ketone bodies.

The development of metabolic acidosis is not distinguishable by clinical observation alone, for acidosis is now a stimulus to hyperventilation and consequent reduction in serum CO_2. As a matter of fact, this reduction serves as a compensatory mechanism to the metabolic acidosis. Hence the child's breathing and the serum CO_2 fails to show which phase of intoxication is present; and, as the urine is not necessarily alkaline during alkalosis, it affords no clarification. Blood pH must be determined for recognition of the state of acidosis. The question is not academic, since therapy is guided by the chemical status of the child.

The ferric chloride test provides presumptive evidence of salicylate poisoning. Normally, ferric chloride gives a Burgundy-red color if aceto-acetic acid is present, but the color does not appear if the specimen is boiled before adding the 10 per cent ferric chloride. If salicylate ion is present, however, the test is positive even after boiling, and gives a violet color. The test is of no quantitative value, however, since a strongly positive reaction may be found in association with relative low blood levels of salicylate. Neither can the test be used to identify aspirin in gastric contents, since the color complex is formed only with free salicylic acid.

Management

The management of salicylism includes close observation and knowledge of the multiple effects of salicylates on the body. These include an increase in body metabolism, lengthened prothrombin time, and blockage of the enzyme systems that normally aid the utilization of ketones. The dehydration, chemical imbalance, and salicylate toxicity combine to impair the peripheral circulation, and an end result is the accumulation of ketone bodies and other acid metabolic products which cannot be handled by the kidney. Thus the original respiratory alkalosis is changed to a metabolic acidosis.

Fever is reduced by cold or tepid sponging; large quantities of parenteral fluids are given to offset dehydration and to augment the elimination of salicylates by increasing urine flow; vitamin K is administered to combat the bleeding tendency due to hypoprothrombinemia. Glucose is needed to overcome ketosis, and electrolyte solutions are required to correct electrolyte imbalance. Sodium bicarbonate or lactate (contraindicated in the early phase when respiratory alkalosis prevails, although there is some doubt that uncomplicated respiratory alkalosis occurs in the very small infant) is given to increase the blood pH, and because salicylates are more promptly excreted in an alkaline urine. Oxygen should be administered to supply the brain, kidneys, and other tissues. Hemodialysis with the artificial kidney and exchange transfusions may be life-saving procedures for methyl salicylate or severe salicylate poisoning. To help the body metabolize salicylate, Diamox (acetazolamide) has recently been used effectively.

. . . . formula dieting is not inherently dangerous. A liquid food mixture is nutritionally equivalent to the same quantity of food in more familiar form. Formula feedings permit exact control of dietary intake—an essential requirement of metabolic studies, and a helpful feature in the initial treatment of obesity. However, liquid mixtures do not have magic properties that allow sedentary and overfed people to lose weight easily and keep it off permanently. Used as an occasional feeding to replace some regular meals, formula feedings are harmless but no more effective than feeding the same number of calories as plain milk, as a serving of fruit as tea and honey, or as any one of countless other simple foods—Dole, V. P.: Editorial: Nine Hundred Calories, New York State J. Med. 61: 1236 (April 15) 1961.

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AUGUST, 1961

THE A.M.A.'S ONE HUNDRED TENTH ANNUAL MEETING

Dr. S. J. L. Blasingame's comprehensive report of the A.M.A.'s One Hundred Tenth annual meeting appears elsewhere in this issue. Only a few editorial comments will be given here.

The registration for this meeting was the highest yet recorded—almost three times that of the Miami meeting last year. The scientific program was well up to the standard set by previous meetings. Members of the House of Delegates, however, had little time to take in the scientific program or the exhibits. The House was in session all day Monday and Wednesday and until 1:00 A.M. Thursday. Those serving on reference committees were busy most of Tuesday.

A record number of resolutions—115—were introduced, in addition to 28 reports. Perhaps the most important action of the House was the approval of the recommendations of the Medical Disciplinary Committee and the direction that its function be assumed by the Judicial Council.

Another important action was the approval of the oral polio vaccine for mass vaccination when it becomes available—meanwhile continuing support of the Salk vaccine.

The door was opened for recognition of osteopaths who base their practice on the scientific principles of regular medicine.

For the Distinguished Service Award, Dr. Walter H. Judd won a large majority of votes over the two other candidates.

The President-Elect, Dr. G. M. Fister, was a dark-horse candidate. It had been assumed that our neighbor, Dr. Julian Price, who has served his full term of 10 years as a member of the Board of Trustees and as Chairman for the last few years, would be elected without opposition—but Dr. Fister, who has also been a trustee for eight years, was elected by a large majority. Although the North Carolina delegates supported Dr. Price, Dr. Fister may be sure that he will have the support of our members in the trying years ahead, and Julian need not be told that we shall continue to hold him in highest esteem and affection.

Dr. Leonard Larson, who was installed as President on Tuesday night in a televised ceremony, won the hearts of North Carolina doctors when he visited us last year in Raleigh. Since his daughter lives in North Carolina, we feel that we have some claim on him.

Dr. Amos Johnson made his debut as North Carolina's fourth delegate and gave a good account of himself. Dr. Schoenheit came for the second time as Dr. Strosnider's alternate. Last year Dr. Strosnider was kept at home by his own illness, this year by that of his wife. Drs. Millard Hill and Elias Faison are, of course, veteran delegates. Dr. Hill was on the important Reference Committee on Legislation and Public Relations.

The New York State Society proved to be most gracious hosts. The dinner dance Monday night was featured by no speeches but by good music, good food, and good fellowship.

One always leaves the annual meeting with the feeling of wonder that so much can be accomplished in so little time. The A.M.A. is indeed the greatest medical show on earth.

* * *

THE 1961 LEGISLATURE

Many citizens have differed with Governor Sanford's appraisal of the 1961 Legislature as being one of the greatest in history. Many believe that the legislature took more backward than forward steps.

A forward step of medical interest was the establishment of a program of inspection for red meats and poultry. Other forward steps of general interest were improvement of our general education program, including a pilot instruction for special instruction of gifted children, and also a program to test the feasibility of recognizing gifted teachers. Another forward step was to stop diverting highway funds for other purposes, such as support of prisons.

Among the backward steps was the defeat of three of four proposals for safety legislation: the bill to require mechanical inspection, a bill permitting chemical tests for alcohol concentration to be used on persons suspected of drunken driving, and a bill to set up penalties for driving under the influence of drugs. The only one of Commissioner Ed Scheidt's recommendations that passed both the Senate and the House was the bill to bolster the driver point system program.

From the medical standpoint, the longest backward step was the defeat of the bill to take advantage of the Kerr-Mills Act. This bill passed the House, but was killed in the Senate. The reason given for killing the bill was really an insult to the medical profession—that the medical aid plan would be dominated by the medical profession. As the *Winston-Salem Journal* commented editorially, "it still seems a shame to have let these funds go to waste, when the medical needs of the aged are so urgent and when for so little North Carolina could have obtained so much."

While the redistricting of the state in order to eliminate one of its present 12 seats in Congress is not a medical matter, doctors are and should be good enough citizens to resent the gerrymandering tactics used in order to pit one of the most popular Democratic Representatives against the sole Republican Representative. It is quite possible that this obviously partisan maneuver will backfire, for almost certainly many voters who usually vote the Democratic ticket will have enough sense of fair play to cross the party line.

Although the rural population of North Carolina is chiefly in the eastern half of the state, the Piedmont section was penalized by the gerrymandering tactics. It is only fair to say, however, that the rural population of the west as well as of the east combined against the urban population. As a result, our state is less democratic in the broad sense of the term than it should be.

A review of the 1961 Legislature shows that all four of the safety measures proposed by Ed Scheidt passed the Senate, but three of them were killed in the House. On the other hand, the Kerr-Mills Act was approved by the House but was killed in the Senate. This observation raises the question if it might not be a good idea to adopt the policy of some of the midwestern states and have a single legislative body instead of two.

The 1961 Legislature achieved one "first." For the first time in our state's history, North Carolina's budget passed the billion dollar mark. There are some living who can remember when the United States first had a billion-dollar budget. The Speaker of the House commented that this was fitting, since the United States was a billion-dollar country. Let us hope that North Carolina will prove truly to be a billion-dollar state.

* * *

"DIAGNOSTIC" OR "CLINICAL" RADIOLOGY

The January issue of this Journal published a letter from the American College of Radiology warning our readers against being misled by two organizations promoted by a California internist, Dr. Louis Shattuck Baer. The older organization was given the imposing name, "The American Society of Diagnostic Radiology." The younger was christened, "The American Society of Clinical Radiology." Because renewed efforts are being made to recruit members for the Society of Clinical Radiology, it is in order to remind our readers again of the nature of this organization.

So far as can be learned, the only qualification required for membership in either organization is ten dollars—and possibly the possession of an x-ray machine. In contrast to these loose standards for membership, the American College of Radiology requires high standards of proficiency in the practice of radiology. These standards are to assure the American public of the best practice of radiology. Anyone who is properly qualified can become a member of the American College of Radiology. Circumvention of these criteria by forming organizations which do not require such training for membership will undermine the best practice of medicine. Membership in them should be avoided.

* * *

AS OTHERS SEE US

One of the most frequently terms used nowadays is "image." Much has been written both in medical and nonmedical publications about the present-day image of the doctor. *New York State Journal of Medicine* for July 1 has such a pertinent, succinct discussion of "Our Corporate Image" that we are reproducing it for the benefit of our own readers.

One of the preoccupations of our times is to view our corporate image. None escape—our nation, our cities, and our businesses and professions are all under scrutiny in this regard. There are serious lessons to be learned from this modern type of narcissism.

A recent private poll by the editor of one of our large daily papers in this State asked the question, "What do you think of doctors?" The editor asked this question personally of random subscribers and friends.

Surprisingly enough, criticism was directed not particularly to the actions or inactions of organized medicine but to the individual doctor's conduct of his practice. Almost universal were the complaints of lack of a warm, understanding contact with the physician, wholesale referral to specialists, particularly for simple, uncomplicated conditions, and high costs, including hospitalization and drug costs which are largely out of the control of the doctor.

Numerous were the complaints of inability to get the doctor "when I want him." This was not a night call problem but one which happened during the business hours of the day. "The doctor is not punctual" was often heard. Sad to relate, there was some criticism of an occasional one of our own who loaded on an extra fee to insurance service benefits which had been firmly agreed on.

There was much praise too for skill and competence. The people like and admire *what* we do, but they are less than enthusiastic about *how* we do it.

The corporate image is the sum total of the individual images. We will *look* better when we *are* better.

Tightening up in extravagances in hospitalization and drug prescribing, closer attention to our patients and a human, holistic approach to their problems, and prudence in referral would please our patients and might not do us a bit of harm as we face the winds of change.

* * *

PROPOSED INCREASE OF TAX ON TRUCKS

Regardless of one's political views, anyone who has thought seriously about the matter must admit that President Kennedy's proposal to increase sharply the tax on trucks according to their weight is logical. Some years ago a highway engineer stated that the crushing impact of one heavy truck did more damage to our hard-surfaced highways than a thousand passenger cars.

Anyone who has driven behind one of the freight cars on rubber wheels that are now so numerous needs no degree in engineering to realize that this estimate was not unreasonable, and that the Behemoths of the highway should bear a more equitable share of the upkeep of our highway system.

Committees & Organizations

Schedule of Committee & Commission Appointments, 1961-1962

NOTE: The Committees listed herein have been authorized by President Claude B. Squires, and/or are required under the Constitution and By-Laws.

Particular note should be taken of the authorization of the House of Delegates of a Commission form of organizational activity and that all Committees, excepting Committee on Nomination, Committee on Negotiation, and Committee on Grievances, are segregated under the respective Commission in which the function of the committee logically rests. This will tend to eliminate overlapping and duplication in activity programs and result in coordination of the work of the Society in a manner to lessen the work of the delegates in the Annual Meeting of the House of Delegates.

(The President, Secretary and Executive Director of the Society are ex-officio members of all committees and, along with the Commission Chairman, should receive notice of meetings, agenda and minutes of committee meetings during the activity year.)

I. **ADMINISTRATION COMMISSION**
Wayne J. Benton, M.D., *Chairman*
2320 Battleground Rd. Committee
Greensboro, North Carolina listing

1. **Finance, Committee on (I-1)** #19
Wayne J. Benton, M.D., *Chairman*
2320 Battleground Rd.
Greensboro, North Carolina

2. **Liaison to Study Integration of Negro Physicians into Medical Society of State of North Carolina** #44
Paul F. Whitaker, M.D., *Chairman*
1205 N. Queen St.
Kinston, North Carolina

3. **Trust Study Committee (I-3)** #45
Jesse Caldwell, M.D., *Chairman*
114 West Third Street
Gastonia, North Carolina

II. **ADVISORY AND STUDY COMMISSION**
Jacob H. Shuford, M.D., *Chairman*
7 Main Avenue Place, S. W.
Hickory, North Carolina

1. **Auxiliary Advisory and Archives of Medical Society History, Committee on, (II-1)** #1
Roscoe D. McMillan, M.D., *Chairman*
P. O. Box 232
Red Springs, North Carolina

2. **American Medical Education Foundation, Committee on, (II-2)** #2
Ralph B. Garrison, M.D., *Chairman*
222 N. Main Street
Hamlet, North Carolina

3. **Blue Shield, Committee on (II-3)** #8
Jacob H. Shuford, M.D., *Chairman*
7 Main Avenue Place, S. W.
Hickory, North Carolina

4. **Constitution and By-Laws, Committee on, (II-4)** #13
Roscoe D. McMillan, M.D., *Chairman*
Box 232
Red Springs, North Carolina

5. **Credit Bureau, Committee on Medical, (II-5)** #14
W. Howard Wilson, M.D., *Chairman*
403 Professional Building
Raleigh, North Carolina

6. **Industrial Commission of North Carolina Committee to Work with, (II-7)** #23
Thomas B. Dameron, Jr., M.D., *Chairman*
600 Wade Avenue
Raleigh, North Carolina

7. **Medical Care of Dependents of Members of Armed Forces, (MEDICARE) Committee on, (II-7)** #28
David M. Cogdell, M.D., *Chairman*
911 Hay Street
Fayetteville, North Carolina

8. **Student A.M.A. Chapters, Committee Advisory to, (II-8)** #41
John P. Davis, M.D., *Chairman*
821 Nissen Building
Winston-Salem, North Carolina

9. **Blue Shield Deputation to National Blue Shield (II-9)** #46
Theodore S. Raiford, M.D., *Chairman*
301 Doctors Bldg.
Asheville, North Carolina

III. **ANNUAL CONVENTION COMMISSION**
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North Carolina Memorial Hospital
Chapel Hill, North Carolina

1. **Arrangements, (of Facilities Annual Session), Committee on, (III-1)** #4
John S. Rhodes, M.D., *Chairman*
700 West Morgan Street
Raleigh, North Carolina

2. **Audio-Visual Scientific Postgraduate Instruction, Committee on, (III-2)** #5
J. Leonard Goldner, M.D., *Chairman*
Duke Hospital
Durham, North Carolina

3. **Awards, Committee on (III-3)** #6
Lester A. Crowell, Jr., M.D., *Chairman*
South Aspen St.
Lincolnton, North Carolina

4. **Delegates, Committee on Credentials to House of Delegates (III-4)** #15
T. Tilghman Herring, M.D., *Chairman*
Wilson Clinic
Wilson, North Carolina

5. **Exhibits, Committee on Scientific, (III-5)** #17
Vernon H. Youngblood, M. D., *Chairman*
609 Kannapolis Highway
Concord, North Carolina

6. **Golf Tournament, Committee on Medical (III-6)** #21
Charles W. Styron, M.D., *Chairman*
615 St. Mary's St.
Raleigh, North Carolina

7. **Scientific Works, Committee on (III-7)** #7
Kenneth M. Brinkhous, M.D., *Chairman*
University of N. C.
Chapel Hill, North Carolina

IV. **PROFESSIONAL SERVICE COMMISSION**
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Raleigh, North Carolina

1. **Emergency Medical and Military Service Committee on (IV-1)** #16
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Raleigh, North Carolina

2. **Eye Care and Eye Bank, Committee on (IV-2)** #18
George T. Noel, M.D., *Chairman*
211 Raleigh Building
Kannapolis, North Carolina

3. **Insurances, Committee on, (IV-3)** #24
Joseph W. Hooper, Jr., M.D., *Chairman*
410 North 11th Street
Wilmington, North Carolina

4. **Necrology, Committee on, (IV-4)** #30
Charles H. Pugh, M.D., *Chairman*
Box 527
Gastonia, North Carolina

5. **Nursing, Committee of Physicians on, (IV-5)** #33
Fred C. Hubbard, M.D., *Chairman*
408 8th Street
North Wilkesboro, North Carolina

6. **Postgraduate Medical Study, Committee on (IV-6)** #35
Wm. P. Richardson, M.D., *Chairman*
N. C. Memorial Hospital
Chapel Hill, North Carolina

V. **PUBLIC RELATIONS COMMISSION**
Hubert McN. Poteat, Jr., M.D., *Chairman*
713 Wilkins Street
Smithfield, North Carolina

1. **Hospital and Professional Relations and Liaison to North Carolina Hospital Association, Committee on (V-1)** #22
Theodore H. Mees, M.D., *Chairman*
501 West 27th Street
Lumberton, North Carolina

2. **Legislation, Committee on, (V-2)** #25
Edgar T. Beddingfield, Jr., M.D., *Chairman*
P. O. Box 137
Stantonsburg, North Carolina

3. **Medical-Legal Committee (V-3)** #27
Julius A. Howell, M.D., *Chairman*
Bowman Gray School of Medicine
Winston-Salem, North Carolina

4. **Public Relations, Committee on (V-4)** #37
David G. Welton, M. D., *Chairman*
403 N. Tryon Street
Charlotte, North Carolina

5. **Rural Health and General Practitioner Award, Committee on (V-5)** #39
Philip E. Dewees, M.D., *Chairman*
Box 27
Sylva, North Carolina

6. **Insurance Industry Liaison Committee** #43
Frank W. Jones, M.D., *Chairman*
Catawba Hospital
Newton, North Carolina

VI. **PUBLIC SERVICE COMMISSION**
Allyn B. Choate, M.D., *Chairman*
1012 Kings Drive
Charlotte, North Carolina

1. **Anesthesia Study, Committee on, (VI-1)** #3
David A. Davis, M.D., *Chairman*
North Carolina Memorial Hospital
Chapel Hill, North Carolina

2. **Board of Public Welfare of North Carolina, Committee Advisory to, (VI-2)** #9
J. Samuel Holbrook, M.D., *Chairman*
Davis Hospital
Statesville, North Carolina

3. **Cancer, Committee on, (VI-3)** #10
James F. Marshall, M.D., *Chairman*
310 West 4th Street
Winston-Salem, North Carolina

4. **Child Health, Committee on, (VI-4)** #11
Angus M. McBryde, M.D., *Chairman*
809 West Chapel Hill Street
Durham, North Carolina

5. **Chronic Illness, Tuberculosis and Heart Disease, Committee on, (VI-5)** #12
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Kernodle Clinic
Burlington, North Carolina

6. **Maternal Health, Committee on, (VI-6)** #26
James F. Donnelly, M.D., *Chairman*
State Board of Health
Raleigh, North Carolina

7. **Mental Health, Committee on, (VI-7)** #29
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8. **Occupational Health, Committee on, (VI-8)** #34
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West Main Street
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104 E. Northwood Street
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10. **Rehabilitation Physical, Committee on (VI-10) #38**
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2240 Cloverdale Avenue
Winston-Salem, North Carolina

11. **School Health, Committee on, (VI-11) #40**
Jean D. Craven, M.D., *Chairman*
19 W. 3rd Avenue
Lexington, North Carolina

12. **Veterans Affairs, Committee on, (VI-12) #42**
Samuel L. Elfmon, M.D., *Chairman*
225 Green Street
Fayetteville, North Carolina

VII. **NOMINATIONS, COMMITTEE ON (not a commission constitutionally provided) #32**
Charles M. Norfleet, Jr., M.D., *Chairman*
Bowman Gray
Winston-Salem, North Carolina

VIII. **GRIEVANCES, COMMITTEE ON, (not a commission By-Law provided) #20**
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408 N. 11th Street
Wilmington, North Carolina

IX. **NEGOTIATIONS, COMMITTEE ON, (not a commission By-Law provided) #31**
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Moore County Hospital
Pinehurst, North Carolina

1. **Committee Advisory to the Auxiliary and Archives of Medical Society History (12) II-1**
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Millard B. Bethel, M.D., School of Public Health, Chapel Hill
Francis N. Bowles, M.D., 306 S. Gregson Street, Durham
Ethel May Brownsberger, M.D., 75 Hendersonville Road, Biltmore
Powell G. Fox, Sr., M.D., 1110 Wake Forest Road, Raleigh
Wingate M. Johnson, M.D., 300 South Hawthorne Road, Winston-Salem
Ruth Leonard, M.D., 106 W. 7th Street, Charlotte 2
George T. Noel, M.D., 211 Raleigh Building, Kannapolis
Zack D. Owens, M.D., Medical Building, Elizabeth City
Rose Pully, M.D., 1007½ N. College Street, Kinston
Paul F. Whitaker, M.D., 1205 N. Queen Street, Kinston

2. **Committee on American Medical Education Foundation (AMEF) (5) II-2**
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William Pettway J. Peete, M.D., Duke Hospital, Durham
Harry B. Underwood, M.D., 709 W. End Avenue, Statesville

3. **Committee on Anesthesia Study Commission (11) VI-1**
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John R. Hoskins, III, M.D., 203 Doctors Bldg., Asheville
John C. Montgomery, M.D., 1400 Scott Avenue, Charlotte
Will Camp Sealy, M.D., Duke Hospital, Durham
Charles R. Stephen, M.D., Box 3535, Duke Hospital, Durham
Thomas B. Wilson, M.D., Rex Hospital Laboratory, Raleigh

4. **Committee on Arrangements (3) (plus 2 consultants) III-1**
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Hugh F. McManus, Jr., M.D., 722 St. Mary's Street, Raleigh
Milard D. Hill, M.D., Consultant, 15 W. Hargett Street, Raleigh
George C. Mackie, M.D., Consultant, Box 72, Wake Forest

5. **Committee on Scientific Audio-Visual Postgraduate Instruction (9) III-2**
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John C. Grier, Jr., M.D., Carthage Road, Pinehurst
William P. Richardson, M.D., N. C. Memorial Hospital, Chapel Hill
H. Frank Starr, M.D., Pilot Life Insurance Company, Greensboro
Frederick Harvey Taylor, M.D., 1012 Kings Drive, Charlotte
J. O. Williams, M.D., Cabarrus Memorial Hospital, Concord
George T. Wolff, M.D., 1311 N. Elm Street, Greensboro

6. **Committee on Scientific Awards (9 plus 1 consultant) III-3**

Lester A. Crowell, Jr., M.D., *Chairman,* (1964), South Aspen Street, Lincolnton
Alton J. Coppridge, M.D. (1964), 1200 Broad Street, Durham
Thomas A. Henson, M.D., (1963), 369 N. Elm Street, Greensboro
Joseph M. Hitch, M.D., (1962), 415 Professional Bldg., Raleigh
Livingstone Johnson, M.D., (1964), 408 W. Warren Street, Shelby
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Vernon W. Taylor, Jr., M.D., (1962), 111 E. Main Street, Elkin
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Mr. Emory Hunt, Consultant, University of North Carolina, Chapel Hill

7. **Committee on Scientific Works (5 plus Section Chairmen as Consultants) III-7**

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Hubert McN. Poteat, Jr., M.D., 713 Wilkins Street, Smithfield
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Joseph S. Hiatt, Jr., M.D., (Internal Medicine), 208 S. W. Broad Street, Southern Pines
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Jacob Koomen, M.D., (Pub. Health & Ed.),
Owen W. Doyle, M.D., (Radiology), 1013 Professional Village, Greensboro
State Board of Health, Raleigh
Thad J. Barringer, M.D., (N&P), Route 6, Leadmine Road, Raleigh
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Bill Joe Swan, M.D., (Anesthesia), 895 Arbor Lane, Concord
Wayne S. Montgomery, M.D., (Ortho & Traumatology), 108 Doctors Bldg., Asheville
Mr. Mike Barringer, (Student AMA Chapters), 2415 Jefferson Avenue, Winston-Salem

8. **Committee on Blue Shield (9) II-3**

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William J. Cromartie, M.D., (1963), UNC School of Medicine, Chapel Hill
Willard C. Goley, M.D., (1962), 214 N. Market Street, Graham
John R. Hoskins, III, M.D., (1963), 203 Doctors Bldg., Asheville
Frederick C. Hubbard, M.D., (1964), 408 8th Street, North Wilkesboro
E. Eugene Menefee, Jr., M.D., (1963), Duke Hospital, Durham
John W. Morris, M.D., (1962), 1707 Arendell Street, Morehead City
Walter T. Tice, M.D., (1964), 624 Quaker Lane, High Point

9. **Committee Advisory to North Carolina State Board of Public Welfare (9) VI-2**

J. Samuel Holbrook, M.D., *Chairman,* Davis Hospital, Statesville
J. Street Brewer, M.D., Box 98, Roseboro
Allyn B. Choate, M.D., 1012 Kings Drive, Charlotte
Lucile W. Hutaff, M.D., Bowman Gray, Winston-Salem
J. Kempton Jones, M.D., 1001 S. Hamilton Road, Chapel Hill
Benjamin H. Kendall, M.D., Shelby Medical Center, Shelby
William W. Noel, M.D., Professional Bldg., Henderson
Logan T. Robertson, M.D., 170 Woodfin Street, Asheville
Wm. Raney Stanford, M.D., 111 Corcoran Street, Durham

10. **Committee on Cancer (12) (Legal—1 each Congressional District) VI-3**

James F. Marshall, M.D., *Chairman,* (5th), 310 W. 4th, Street, Winston-Salem
William H. Bell, Jr., M.D., (3rd), P. O. Box 1580, New Bern
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Arthur B. Bradsher, M.D., (6th), 1200 Broad Street, Durham
Charles I. Harris, Jr., M.D., (1st), Martin General Hospital, Williamston
Harry V. Hendrick, M.D., (11th), Rutherford Hospital, Rutherfordton
Harold A. Peck, M.D., (8th), Moore Memorial Hospital, Pinehurst
David L. Pressly, M.D., (9th), 1025 Davie Street, Statesville
Samuel L. Parker, Jr., M.D., (2nd), Kinston Clinic, Kinston
Hubert McN. Poteat, Jr., M.D., (4th), 713 Wilkins Street, Smithfield

D. Ernest Ward, Jr., M.D., (7th), 304 Medical Arts Bldg., Lumberton
William L. Williams, Jr., M.D., (10th), Mercy Hospital, Charlotte

11. **Committee on Child Health (9) VI-4**
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Harrie R. Chamberlin, M.D., UNC School of Medicine, Chapel Hill
Katherine Anderson, M.D., 138 N. Hawthorne Rd., Winston-Salem
John W. Nance, M.D., 401 Cooper Drive, Clinton
Wm. H. Patton, Jr., M.D., 305 College Street, Morganton
Robert L. Vann, M.D., 301 Miller Street, Winston-Salem
Robert F. Poole, Jr., M.D., 817 Hillsboro Street, Raleigh

12. **Committee on Chronic Illness, Including Tuberculosis and Heart Disease (16) VI-5**
John R. Kernodle, M.D., *Chairman*, Kernodle Clinic, Burlington
Stephen R. Bartlett, Jr., M.D., 1001 E. 4th Street, Greenville
Robert H. Dovenmuehle, M.D., Duke Hospital, Durham
O. David Garvin, M.D., Health Department, Chapel Hill
Robert A. Gregg, M.D., Central Convalescent Hospital, Greensboro
Emery T. Kraycirik, M.D., Box 1153, Burlington
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Thomas R. Nichols, M.D., 206 N. Sterling Street, Morganton
Elbert L. Persons, M.D., Duke Hospital, Durham
Richard C. Proctor, M.D., Bowman Gray, Winston-Salem
John L. Shirey, M.D., 1 Battle Square, Asheville
George L. Verdone, M.D., 1012 Kings Drive, Charlotte
Donald D. Weir, M.D., N. C. Memorial Hospital, Chapel Hill
William H. Flythe, M.D., 624 Quaker Lane, High Point

13. **Committee on Constitution & By-Laws (5) II-4**
Roscoe D. McMillan, M.D., *Chairman*, Box 232, Red Springs
Millard D. Hill, M.D., 15 W. Hargett Street, Raleigh
G. Westbrook Murphy, M.D., 103 Doctors Bldg., Asheville
Edward W. Schoenheit, M.D., 46 Haywood Street, Asheville
Louis deS. Shaffner, M.D., 300 S. Hawthrone Road, Winston-Salem

14. **Committee on Medical Credit Bureaus (7) II-5**
W. Howard Wilson, M.D., *Chairman*, 403 Professional Bldg., Raleigh
Paul McN. Deaton, M.D., 766 Hartness Road, Statesville
Robert M. Fales, M.D., 913 Murchison Bldg., Wilmington
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John R. Hoskins, III, M.D., 203 Doctors Bldg., Asheville
Ross S. McElwee, Jr., M.D., 1340 Romany Road, Charlotte
Ralph J. Sykes, M.D., 205 Rawley Avenue, Mt. Airy

15. **Committee on Credentials of Delegates to House of Delegates (5) III-4**
T. Tilghman Herring, M.D., *Chairman*, Wilson Clinic, Wilson
John F. Ditunno, Jr., M.D., Hot Springs
James E. Hemphill, M.D., 1012 Kings Drive, Charlotte
Robert M. Whitley, M.D., 144 Coast Line Street, Rocky Mount
Charles B. Wilkerson, M.D., 100 S. Boylan Avenue, Raleigh

16. **Committee on Emergency Medical and Military Service (7) IV-1**
George W. Paschal, Jr., M.D., *Chairman*, 1110 Wake Forest Road, Raleigh
Chauncey L. Royster, M.D., Co-Chairman, 707 W. Morgan Street, Raleigh
Charles E. Cloninger, M.D., 305 E. 1st Street, Conover
Charles Davant, Jr., M.D., Blowing Rock Hospital, Blowing Rock
J. Kingsley MacDonald, M.D., 1524 Harding Place, Charlotte
H. Mack Pickard, M.D., 7 N. 17th Street, Wilmington
George A. Watson, M.D., 306 S. Gregson Street, Durham

17. **Committee on Scientific Exhibits (7) III-5**
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W. Reece Berryhill, M.D., UNC School of Medicine, Chapel Hill
William H. Boyce, M.D., Bowman Gray, Winston-Salem
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George Preston Nowlin, M.D., 1012 Kings Drive, Charlotte
Max P. Rogers, M.D., 624 Quaker Lane, High Point

M. Barnes Woodhall, M.D., Duke Medical Center, Durham

18. **Committee on Eye Care and Eye Bank (8) IV-2**

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Henry H. Briggs, Jr., M.D., 416 Doctors Bldg., Asheville
George M. Cooper, Jr., M.D., 2111 Clark Avenue, Raleigh
George A. Levi, M.D., P. O. Box 3364, Fayetteville
Marvin N. Lymberis, M.D., 106 W. 7th Street, Charlotte 2
Edward E. Moore, M.D., 706 Flatiron Bldg., Asheville
Carl N. Patterson, M.D., 1110 W. Main Street, Durham
J. David Stratton, M.D., 1012 Kings Drive, Charlotte

19. **Committee on Finance (3) (plus 11 consultants) I-1**

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Elias S. Faison, M.D., 1012 Kings Drive, Charlotte
W. Walton Kitchin, M.D., Sampson County Memorial Hospital, Clinton
Consultants: Graham B. Barefoot, M.D., Box 1198, Wilmington
Howard H. Bradshaw, M.D., Bowman Gray, Winston-Salem
Allyn B. Choate, M.D., 1012 Kings Drive, Charlotte
Paul McN. Deaton, M.D., 766 Hartness Rd., Statesville
Isaac E. Harris, Jr., M.D., 1200 Broad Street, Durham
John W. Ormand, Sr., M.D., Box 397, Monroe
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Edward W. Schoenheit, M.D., 46 Haywood Street, Asheville
O. Norris Smith, M.D., 1019 Professional Village, Greensboro
John C. Tayloe, M.D., 120 Washington Street, Washington
Heyward C. Thompson, M.D., Box 1202, 314 S. Washington Street, Shelby

20. **Committee on Grievances (5) (1st Five Past Presidents) VIII-0**

Donald B. Koonce, M.D., *Chairman*, 408 N. 11th Street, Wilmington
Amos N. Johnson, M.D., Secretary, Garland
John C. Reece, M.D., Grace Hospital, Morganton
Lenox D. Baker, M.D., Duke Hospital, Durham
Edward W. Schoenheit, M.D., 16 Haywood Street, Asheville

21. **Committee on Medical Golf Tournament (3) III-6**

Charles W. Styron, M.D., *Chairman*, 615 St. Mary's Street, Raleigh
James H. Manly, Jr., M.D., 2007 Clark Avenue, Raleigh
L. Gordon Sinclair, M.D., 704 W. Jones Street, Raleigh

22. **Committee on Hospital and Professional Relations and Liaison to North Carolina Hospital Association (10) V-1**

Theodore H. Mees, M.D., *Chairman*, (5th), 501 W. 27th Street, Lumberton
Frederick P. Brooks, M.D., (2nd), 525 Evans Street, Greenville
Quinton E. Cooke, M.D., (1st), 209 E. Main Street, Murfreesboro
Paul McN. Deaton, M.D., (9th), 766 Hartness Rd., Statesville
John Tyler Dees, M.D., (3rd), Box 248, Burgaw
Frederick C. Hubbard, M.D., (8th), Box 30, North Wilkesboro
H. Lee Large, Jr., M.D., (7th), Presbyterian Hospital, Charlotte
Arthur H. London, Jr., M.D., (6th), 306 S. Gregson Street, Durham
James S. Raper, M.D., (10th), Doctors Bldg., Asheville
Jack W. Wilkerson, M.D., (4th), Community Clinic, Stantonsburg

23. **Committee to Work with North Carolina Industrial (7) II-6**

Thomas B. Dameron, Jr., M.D., *Chairman*, 600 Wade Avenue, Raleigh
R. W. Coonrad, M.D., Broad & Englewood Streets, Durham
Joe Walton Frazer, Jr., M.D., 838 N. Elm Street, Greensboro
James S. Mitchener, Jr., M.D., Scotland County Memorial Hospital, Laurinburg
Malory A. Pittman, M.D., Wilson Clinic, Wilson
William Reid Pitts, M.D., 1012 Kings Drive, Charlotte
Charles T. Wilkinson, M.D., 205 Waite Street, Wake Forest

24. **Committee on Insurance (8) IV-3**

Joseph W. Hooper, Jr., M.D., *Chairman*, 410 N. 11th Street, Wilmington
Robert H. Brashear, Jr., M.D., N. C. Memorial Hospital, Chapel Hill
John C. Burwell, Jr., M.D., 1026 Professional Village, Greensboro
Barry F. Hawkins, M.D., Ardsley Road, Concord
D. Forrest Moore, M.D., P. O. Box 136, Shelby

Charles K. Padgett, M.D., 809 LaFayette Street, Shelby
Robert E. Sandy, M.D., Doctors Clinic, Washington
S. Glenn Wilson, M.D., Box 158, Angier

25. Committee on Legislation (3 members plus President & Secretary) (10 Consultants) V-2

Edgar T. Beddingfield, Jr., M.D., *Chairman*, P. O. Box 137, Stantonsburg
Hubert McN. Poteat, Jr., M.D., 713 Wilkins Street, Smithfield
George W. Paschal, Jr., M.D., 1110 Wake Forest Road, Raleigh
Claude B. Squires, M.D., President (Ex Offico), 225 Hawthorne Lane, Charlotte
John S. Rhodes, M.D., Secretary (Ex Officio), 700 W. Morgan Street, Raleigh
Consultants:
  Daniel S. Currie, Jr., M.D., 111 Bradford Ave., Fayetteville
  Joseph S. Holbrook, M.D., Davis Hospital, Statesville
  William E. Keiter, M.D., 400 Glenwood Ave., Kinston
  Donald B. Koonce, M.D., 408 N. 11th Street, Wilmington
  Leslie M. Morris, M.D., Medical Building, Gastonia
  Charles M. Norfleet, Jr., M.D., Bowman Gray, Winston-Salem
  Zack D. Owens, M.D., Medical Building, Elizabeth City
  Robert Stuart Roberson, M.D., 102 Brown Avenue, Hazelwood
  Ben F. Royal, M.D., 900 Shepherd Street, Morehead City
  Thomas B. Dameron, Jr., M.D., 1313 600 Wade Avenue, Raleigh

26. Committee on Maternal Health (14) VI-6

James F. Donnelly, M.D., *Chairman*, (1966), State Board of Health, Raleigh
W. Joseph May, M.D., Secretary, (8th), 121 Professional Bldg., Winston-Salem
Glenn E. Best, M.D., (3rd)-(1966), Main Street, Clinton
Jesse Caldwell, M.D., (7th)-(1967), 114 West Third Street, Gastonia
P. J. McElrath, M.D., (6th)-(1967), 500 St. Mary's Street, Raleigh
Milton S. Clark, M.D., (4th)-(1967), Wachovia Bank Bldg., Goldsboro
W. Otis Duck, M.D., (10th)-(1963), Box 387, Mars Hill
Wm. A. Hoggard, Jr., M.D., (1st)-(1965), 1502 Carolina Avenue, Elizabeth City
Wm. R. Wellborn, Jr., M.D., (9th)-(1964), 222 W. Union Street, Morganton
Frank R. Lock, M.D., (BG)-(1965), 300 S. Hawthorne Rd., Winston-Salem
Hugh A. McAllister, M.D., (5th)-(1965), 27th at Barker Street, Lumberton
Roy T. Parker, M.D., (Duke)-(1966), Box 3517, Duke Hospital, Durham
Robert A. Ross, M.D., (UNC)-(1963), N. C. Memorial Hospital, Chapel Hill
H. Fleming Fuller, M.D., (2nd)-(1963), Kinston Clinic, Kinston

27. Medical-Legal Committee (8) V-3

Julius A. Howell, M.D., *Chairman*, Bowman Gray, Winston-Salem
Theodore S. Raiford, M.D., 301 Doctors Bldg., Asheville
David G. Welton, M.D., 403 N. Tryon Street, Charlotte
John W. Foster, M.D., Veterans Administration, Winston-Salem
Connell G. Garrenton, M.D., Bethel Clinic, Bethel
June U. Gunter, M.D., Watts Hospital, Durham
Bennette B. Pool, M.D., 414 Nissen Bldg., Winston-Salem
Thomas A. Stokes, Jr., M.D., Brighton & Mumford Road, Durham

28. Committee on Medical Care Armed Forces Dependents ("MEDICARE") (15) (plus SubCommittee Consultants—19) II-7

David M. Cogdell, M.D., *Chairman*, 911 Hay Street, Fayetteville
Everett I. Bugg, Jr., M.D., Broad and Englewood Streets, Durham
Jesse Caldwell, M.D., 114 West Third Street, Gastonia
Samuel E. Warshauer, 301 N. 10th Street, Wilmington
Daniel S. Currie, Jr., M.D., 111 Bradford Avenue, Fayetteville
A. Ledyard DeCamp, M.D., 1505 Elizabeth Avenue, Charlotte
Powell G. Fox, Sr., M.D., 1110 Wake Forest Road, Raleigh
Charles Highsmith, M.D., Montgomery Memorial Hospital, Troy
William F. Hollister, M.D., Moore County Hospital, Pinehurst
William Isaac Jones, M.D., 2914 Crosby Street, Charlotte 7
Donald B. Koonce, M.D., 408 N. 11th Street, Wilmington
J. Douglas McRee, M.D., 2109 Clark Avenue, Raleigh
Edwin L. Pierce, M.D., 1110 Wake Forest Road, Raleigh
George A. Watson, M.D., 306 S. Gregson Street, Durham
John L. McCain, M.D., Wilson Clinic, Wilson
A.—General Medicine
  John L. McCain, M.D., *Chairman*, Wilson Clinic, Wilson
  W. R. Stafford, Jr., M.D., 948 Walker Avenue, Greensboro
  Leonard E. Fields, M.D., Box 788, Chapel Hill

Joseph M. Hitch, M.D., 415 Professional Bldg., Raleigh

B.—Radiology

James E. Hamphill, M.D., *Chairman*, 1012 Kings Drive, Charlotte

Joe Lee Frank, Jr., M.D., Roanoke-Chowan Hospital, Ahoskie

C.—Surgery

Wayne H. Stockdale, M.D., *Chairman*, 703 North Street, Smithfield

Howard M. Ausherman, M.D., 200 Hawthorne Lane, Charlotte

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George R. Miller, M.D., 412 Realty Bldg., Gastonia

Guy L. Odom, M.D., Duke Hospital, Fayetteville

C. F. Siewers, M.D., 1110 W. Main Street, Durham

Larry Turner, M.D., 1110 West Main Street, Durham

Frank Edward Altany, M.D., 1012, Kings Drive, Charlotte

D.—Obstetrics & Gynecology

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29. **Committee on Mental Health (15) VI-7**

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William E. Bellamy, Jr., M.D., State Hospital, Raleigh

Wilmer C. Batts, Jr., M.D., 2109 Clark Avenue, Raleigh

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Milton S. Clark, M.D., Wachovia Bank Bldg., Goldsboro

James F. Elliott, M.D., State Hospital, Butner

John W. Ervin, M.D., Box 132, State Hospital, Morganton

John A. Fowler, M.D., 2212 Erwin Road, Durham

Thomas T. Jones, M.D, 604 W. Chapel Hill Street, Durham

Hans Lowenbach, M.D., Duke Hospital, Durham

Phillip G. Nelson, M.D., 1211 Rock Spring Road, Greenville

James T. Proctor, M.D., 428 Ridgefield Road, Chapel Hill

Walter A. Sikes, M.D., State Hospital, Raleigh

Joseph B. Stevens, M.D., 1017 Professional Village, Greensboro

David A. Young, M.D., 714 St. Mary's Street, Raleigh

30. **Committee on Necrology (3) IV-4**

Charles H. Pugh, M.D., *Chairman*, Box 527, Gastonia

John W. Harbison, M.D., 911 Washington Street, Shelby

Ben F. Royal, M.D., Box 628, Morehead City

31. **Committee on Negotiations (3) IX-0**

Wm. F. Hollister, M.D., *Chairman* (Term expires 1967), More County Hospital, Pienhurst

Theodore S. Raiford, M.D., (Term expires 1963), 301 Doctors Bldg., Asheville

Hubert McN. Poteat, Jr., M.D., (Term expires 1965), 713 Wilkins Street, Smithfield

32. **Nominating Committee**

Charles M. Norfleet, Jr., M.D., *Chairman*, (8th), Bowman Gray, Winston-Salem

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Karl B. Pace, M.D., (2nd), Box 620, Greenville

Glenn E. Best, M.D., (3rd), Main Street, Clinton

Malory A. Pittman, M.D., (4th), Wilson Clinic, Wilson

Riley M. Jordan, M.D., (5th), 110 Campus Avenue, Raeford

Lenox D. Baker, M.D., (6th), Duke Hospital, Durham

Wm. F. Eckbert, M.D., (7th), Box 317, Cramerton

Gilbert M. Billings, M.D., (9th), 405 S. Sterling Street, Morganton

John B. Anderson, M.D., (10th), 215 Doctors Bldg., Asheville

33. **Committee of Physicians on Nursing (7) IV-5**

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Harry L. Brockmann, M.D., 624 Quaker Lane, High Point

John L. McCain, M.D., Wilson Clinic, Wilson

J. Samuel Holbrook, M.D., Davis Hospital, Statesville

David T. Tayloe, M.D., 209 N. Washington Street, Washington

Thomas J. Taylor, M.D., 643 Roanoke Avenue, Roanoke Rapids

Nursing Careers—Subcommittee

Andrew J. Crutchfield, M.D., *Chairman*, Suite 93 Professional Bldg., Winston-Salem

Improvement of the Care of the Patient—Subcommittee
Harry L. Brockmann, M.D., *Chairman,* 624 Quaker, Lane, High Point
John L. McCain, M.D., Wilson Clinic, Wilson

34. **Committee on Occupational Health (11) VI-8**
John Moir Hall, M.D., *Chairman,* West Main Street, Elkin
Harry L. Johnson, M.D., Box 530, Elkin
B. Joseph Christian, M.D., 948 Walker Street, Greensboro
Mac Roy Gasque, M.D., Pisgah Forest
J. L. Fritz, M.D., Box 990, Asheboro
Clifton G. Payne, M.D., American Tobacco Co., Reidsville
James F. McMillan, M.D., 308 N. 3rd Street, Wilmington
James Kent Rhodes, M.D., 307 Woodburn Road, Raleigh
William P. Richardson, M.D., N. C. Memorial Hosp., Box 758, Chapel Hill
Logan T. Robertson, M.D., 170 Woodfin Street, Asheville
W. B. Townsend, M.D., Box 420, Charlotte

35. **Committee on Postgraduate Medical Study (7) IV-6**
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Ewald W. Busse, M.D., Duke Hospital, Durham
W. Otis Duck, M.D., Box 387, Mars Hill
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Robert L. McMillan, M.D., 2240 Cloverdale Avenue, Winston-Salem
Frank R. Reynolds, M.D., 1613 Dock Street, Wilmington
George T. Wolff, M.D., 1311 N. Elm Street, Greensboro

36. **Committee on Poliomyelitis (14) VI-9**
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Edward P. Benbow, Jr., M.D., 104 E. Northwood Street, Greensboro
Charles R. Bugg, M.D., 627 W. Jones Street, Raleigh
Ralph B. Garrison, M.D., P. O. Box 71, Hamlet
Wm. F. Harrell, Jr., M.D., Guaranty Bank Bldg., Elizabeth City
Julian E. J. Jacobs, M.D., 1822 Brunswick Avenue, Charlotte 7
Richard S. Kelly, M.D., 1606 Morganton Road, Fayetteville
Donald B. Koonce, M.D., 408 N. 11th Street, Wilmington
Robert C. Pope, M.D., Wilson Clinic, Wilson
Lawrence E. Metcalf, M.D., 314 Doctors Bldg., Asheville
William A. Sams, M.D., Box BB, Marshall
John W. Varner, M.D., Box 522, Lexington
Robert F. Young, M.D., Halifax County Health Dept., Halifax

37. **Committee on Public Relations (3)—(plus 8 Consultants) V-4**
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Courtney D. Egerton, M.D., (6th) (1963), 714 St. Mary's Street, Raleigh
Edgar T. Beddingfield, Jr., M.D., (4th) (1962), P. O. Box 137, Stantonsburg
Ralph B. Garrison, M.D., (5th) (1964), P. O. Box 71, Hamlet
Consultants:
R. D. Minges, M.D., (2nd), Rt. 1 Box 6B, Greenville
Glenn E. Best, M.D., (3rd), Main Street, Clinton
William H. Burch, M.D., (10th), Bat Cave
J. Samuel Holbrook, M.D., (9th), Davis Hospital, Statesville
John R. Bender, M.D., (8th), 1401 S. Hawthorne Road, Winston-Salem
William A. Hoggard, M.D., (1st), 1502 Carolina Avenue, Elizabeth City
Philip Naumoff, M.D., 1012 Kings Drive, Charlotte

38. **Committee on Physical Rehabilitation (8) VI-10**
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Charles H. Ashford, M.D., 603 Pollock Street, New Bern
F. P. Dale, M.D., Kinston Clinic, Kinston
J. Leonard Goldner, M.D., Duke Hospital, Durham
Walter S. Hunt, Jr., M.D., 600 Wade Avenue, Raleigh
John Hays Rosser, M.D., 222 N. Center Street, Statesville
Marion B. Pate, Jr., M.D., 123 N. Second Street, St. Pauls
George H. Wadsworth, M.D., 405 Colony Avenue, Ashokie

39. **Committee on Rural Health and General Practitioner Award (9) V-5**
Philip E. Dewees, M.D., *Chairman,* Box 217, Sylva
Spencer A. Bell, M.D., Hamptonville
Edward L. Boyette, M.D., Kenansville
Francis M. Carroll, M.D., 722 North Brown Street, Chadbourn
Wm. F. Eckbert, M.D., Box 317, Cramerton
R. Vernon Jeter, M.D., Plymouth Clinic, Plymouth
John W. Neal, M.D., Main Street, Gibson
R. D. Richards, M.D., Rt. 1, Wilson
Charles T. Wilkinson, M.D., 205 Waite Street, Wake Forest

40. **Committee on School Health and State Coordinating Service (9) VI-11**

Jean Davidson Craven, M.D., *Chairman*, 19 W. 3rd Avenue, Lexington
Irma C. Henderson Smathers, M.D., 1295 Merrimon Avenue, Asheville
Bruce B. Blackmon, M.D., Buies Creek
Joseph S. Bower, M.D., Box 12, Pink Hill
William C. Hunter, M.D., 103 Pine Street, Wilson
Floyd L. Knight, M.D., 103 Hillcrest Drive, Sanford
R. T. Lucas, Jr., M.D., 1350 Kings Drive, Charlotte 7
James W. Rose, M.D., Drawer C., Pikeville
Wm. T. Rainey, Sr., M.D., Highsmith Hospital, Fayetteville

41. **Committee Advisory to Student A.M.A. Chapters in North Carolina (8) II-8**
John P. Davis, M.D., *Chairman*, 821 Nissen Bldg., Winston-Salem
Arthur H. London, Jr., M.D., 306 S. Gregson Street, Durham
Robert L. McMillan, M.D., (BG), Bowman Gray, Winston-Salem
Wm. P. J. Peete, M.D., (Duke), Duke Hospital, Durham
Louis C. Roberts, M.D., 1200 Broad Street, Durham
Robert A. Ross, M.D., (UNC), N. C. Memorial Hospital, Chapel Hill
Claude T. Whittington, M.D., 108 E. Northwood Street, Greensboro
Thomas C. Worth, M.D., Rex Hospital, Raleigh

42. **Committee on Veterans Affairs (9) VI-12**
Samuel L. Elfmon, M.D., *Chairman*, 225 Green Street, Fayetteville
Vernon L. Andrews, M.D., Box 407, Mt. Gilead
Wilmer C. Betts, M.D., 2109 Clark Avenue, Raleigh
H. Francis Forsyth, M.D., Bowman Gray, Winston-Salem
David L. Phillips, M.D., 110 Oak Avenue, Spruce Pine
Robert L. Phillips, M.D., 153 Bishop Street, Greensboro
James E. Davis, M.D., 1200 Broad Street, Durham
John T. Sessions, Jr., M.D., UNC Dept. of Medicine, Chapel Hill

43. **Insurance Industry Liaison Committee (10) V-6**
Frank W. Jones, M.D., *Chairman*, Catawba Hospital, Newton
Jack E. Mohr, M.D., Co-Chairman, Medical Arts Bldg., Lumberton
Gilbert M. Billings, M.D., 405 S. Sterling Street, Morganton
Andrew J. Dickerson, M.D., 1600 N. Main Street, Waynesville
Archie Y. Eagles, M.D., 407 Colony Avenue, Ahoskie
Powell G. Fox, Jr., M.D., 1110 Wake Forest Road, Raleigh
Cleon W. Goodwin, M.D., Wilson Clinic, Wilson
Charles I. Harris, Jr., M.D., Martin General Hospital, Williamston
Barry F. Hawkins, M.D., Ardsley Road, Concord
Robert H. Shackelford, M.D., 115 W. Main Street, Mt. Olive

44. **Committee Liaison to Study Integration of Negro Physicians into Medical Society of the State of North Carolina (9) I-2**
Paul F. Whitaker, M.D., *Chairman*, 1205 N. Queen Street, Kinston
J. Street Brewer, M.D., P. O. Box 98, Roseboro
James E. Hemphill, M.D., 1012 Kings Drive, Charlotte 7
Joseph W. Hooper, Jr., M.D., 410 N. 11th Street, Wilmington
Jack Hughes, M.D., 1200 Broad Street, Durham
Amos N. Johnson, M.D., Garland
Henry B. Perry, Jr., M.D., 344 North Elm Street, Greensboro
James P. Rousseau, M.D., 1014 West 5th Street, Winston-Salem
Ben F. Royal, M.D., 900 Shepherd Street, Morehead City

45. **Trust Study Committee (6) I-3**
Jesse Caldwell, M.D., *Chairman*, 114 West Third Street, Gastonia
Wayne J. Benton, M.D., 2320 Battleground Road, Greensboro
Elias S. Faison, M.D., 1012 Kings Drive, Charlotte
Paul W. Johnson, M.D., 216 Professional Bldg., Winston-Salem
W. Walton Kitchin, M.D., Sampson County Memorial Hospital, Clinton
A. Hewitt Rose, Jr., M.D., 2009 Clark Avenue, Raleigh

46. **Blue Shield Deputation to National Blue Shield (3) II-9**
Theodore S. Raiford, M.D., *Chairman*, 301 Doctors Bldg., Asheville
Jacob H. Shuford, M.D., 7 Main Avenue Place, S. W., Hickory
Edgar T. Beddingfield, Jr., M.D., P. O. Box 137, Stantonsburg

Bulletin Board

COMING MEETINGS

Cape Fear Valley Hospital, Fourth Annual Symposium—Cape Fear Valley Hospital, Fayetteville, September 21.

Forsyth County Heart Association, Twelfth Annual Symposium—Hotel Robert E. Lee, Winston-Salem, September 22.

Symposium on The Commonwealth of Children—Duke University, Durham, October 4-8.

American College of Physicians, Postgraduate Course No. 2, The Physiologic Basis of Internal Medicine—Duke University Medical Center, Durham, October 9-13.

North Carolina Fifth District Medical Society Meeting—Carolina Hotel, Pinehurst, October 11-12.

North Carolina Division, American Cancer Society Meeting — Hotel Sir Walter, Raleigh, October 14-15.

Thirteenth Postgraduate Assembly in Endocrinology and Metabolism, the Endocrine Society and the National Institutes of Health, Bethesda, Maryland, October 2-6.

South Carolina State Orthopedic Meeting—William Hilton Inn, Hilton Head Island, South Carolina, September 15-17.

American Fracture Association, Annual Meeting—Shoreham Hotel, Washington, D. C., September 17-22.

Tennessee Valley Medical Assembly—Read House, Chattanooga, September 25-26; Joseph W. Graves, 109 Medical Arts Building, Chattanooga, Chairman.

A.M.A. Twenty-first National Congress on Occupational Health—Denver, Colorado, October 2-4.

A.M.A. Department of Investigation, First National Congress on Medical Quackery—Washington, D. C., October 6-7.

American Rhinologic Society, Seventh Annual Meeting—Belmont Hotel, Chicago, October 7.

American Association of Medical Colleges, Seventy-second Annual Meeting—Queen Elizabeth Hotel, Montreal, Canada, November 13-15.

NEW MEMBERS OF THE STATE SOCIETY

Dr. Arch Thomas McCoy, 2404 Vinewood Drive, Durham; Dr. Jack Erwyn Dunlap, 216 East 16th Street, Lumberton; Dr. Devereaux Haigh Lippitt, 708 Broad Street, New Bern; Dr. Dankwart Eugen Harald Rodler, New Bern Doctors Building, New Bern; Dr. Ursula Mary Anderson, 619 Ralph Drive, Raleigh; Dr. Edward Larkin Groover, Bertie County Memorial Hospital, Windsor; Dr. Dennis William Brosnan, III, Doctors Building, Asheville; Dr. Raeford Theodore Pugh, 437 East 12th Street, Washington; Dr. Charles Warren Williams, 1218 Beatties Ford Road, Charlotte; Dr. Albert McCray Jones, 408 East 12th Street, Washington; Dr. Joseph Budinot Johnston, Jr., 180 Monroe St., Herndon, Virginia; Dr. Robert Bennett Hodgson, 611 Maple Avenue, Reidsville.

NEWS NOTES FROM THE BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

Dr. Camillo Artom, professor of biochemistry, left June 29, for his native country, Italy, where he will visit in departments of biochemistry at the Universities of Milan, Rome, Florence and Padua. In August, he will fly to Moscow, USSR, to participate as a discussion leader at the Fifth International Congress of Biochemistry. He will also present a paper entitled "Further Studies on the Formation of Phosphatidyl Ethanolamine in Liver Preparations." The paper was prepared by Dr. Artom and Dr. Hugh B. Lofland Jr., associate professor of biochemistry.

* * *

Dr. Richard L. Burt, professor of obstetrics and gynecology, has been elected to membership in the American Gynecological Society. Membership in the society is limited to 100. There are only three other members from North Carolina. One of these is Dr. Frank R. Lock, professor of obstetrics and gynecology, who is treasurer of the society.

* * *

Dr. Frank R. Johnston, assistant professor of surgery, has been elected to membership in the American Association for Thoracic Surgery.

* * *

Dr. Norman M. Sulkin, William Neal Reynolds Professor of Anatomy, has been appointed to the American Biological Research Committee of the International Association of Gerontology. One of the primary functions of the committee will be to plan the program for the Sixth International Congress of Gerontology to be held in Copenhagen in 1963.

* * *

Dr. Joseph E. Whitley, instructor in radiology, has been awarded the James Picker Foundation's advanced fellowship in academic radiology by the National Academy of Sciences. He will study angiography and angiocardiography during a six-month tenure at the Karolinska Institute at Stockholm. He will then return to the United States for a six-month appointment at the Massachusetts Institute of Technology, where he will study radiology instrumentation and research methods. At the same time, he will be associated with the Department of Radiology at the Massachusetts General Hospital at Boston.

* * *

Several members of the medical school faculty participated in sessions of the American Medical Association meeting, June 24 to 30, in New York. —Drs. C. Nash Herndon, professor and chairman of the Department of Preventive Medicine and Medical Genetics, and Robert W. Prichard, professor of pathology, participated in the A.M.A. Multiple Discipline Research Forum. Dr. Herndon spoke on "Basic Contributions to Medicine by Research in Genetics" and participated in group discussions which related basic science research to clinical problems. Dr. Prichard presented a paper entitled "Studies on the Atherosclerotic Pigeon." The paper is a summary of the work of the medical school's multidisciplinary study group on atherosclerosis.

—Dr. LeRoy Crandell, associate professor of anesthesiology, spoke on "The Anesthetic Hazards of Patients on Anti-hypertensive Therapy."

—Dr. Frank R. Lock, professor of obstetrics and gynecology, spoke to the Section on Obstetrics and Gynecology on "The Diagnosis of Congenital Anomalies and a Report of the Incidence Following Rubella in Pregnancy." He also participated in a panel discussion on "Prenatal Influences on the Newborn" with a discussion on "Birth Trauma."

An exhibit on gastric cytology, prepared by the medical school's Departments of Pathology and Medical Illustration, won second place in the medical photography division for scientific exhibits at the American Medical Association meeting. The exhibit was one of the two awards given for medical photography. There were 382 exhibits on display.

* * *

The following faculty appointments were announced recently: Dr. D. Louise Odor as assistant professor of anatomy (electron microscopy); Dr. Tom P. Coker as instructor in orthopedics; Dr. Robert C. McKone as instructor in pediatrics; Dr. James Blake Thomas as instructor in anatomy; Drs. Edward S. Carr, David H. Fuller Jr., and Walter A. Sikes as assistant professors of clinical psychiatry; Drs. James L. Cathell and Stanley James Rogers as instructors in clinical psychiatry; and Dr. Charles G. Gunn as lecturer in industrial medicine.

News Notes from the University of North Carolina School of Medicine

Dr. Carl W. Gottschalk, faculty member of the University of North Carolina School of Medicine, has been awarded the lifetime post of Career Investigator by the American Heart Association.

This announcement was made recently by the American and North Carolina Heart Associations and the University of North Carolina.

The Career Investigatorship provides support throughout their professional lives to scientists of outstanding ability and achievement. Recipients are enabled to pursue research of their own choice at any institution they wish, without requirements to publish or time limitations on their projects.

Dr. Gottschalk is studying the pressure and chemical composition of the fluid in the microscopically small tubules of the kidney. By recording the pressure within the kidney tubule and by analyzing fluids they withdraw through the needle, the research workers are assembling facts concerning the manner in which the kidney operates in health and disease. The project is also aimed at revealing more about the relation between the circulatory system and the kidneys.

* * *

Dr. Stuart Willis of Chapel Hill, clinical professor of medicine and superintendent and medical director of the North Carolina Sanatorium System, was recently installed as president of the National Tuberculosis Association at the group's annual meeting in Cincinnati, Ohio.

Dr. Willis is a past president of the American Thoracic Society and the North Carolina Tuberculosis Association. In 1956 he received the Southern Conference Medal in recognition of his service in the cause of tuberculosis control.

* * *

Among those receiving honorary degrees at the University's commencement exercises on June 5 was Dr. Joseph T. Wearn, native North Carolinian who is Dean of the School of Medicine at Western Reserve University in Cleveland, Ohio.

Dr. Cadmus has been director of Memorial Hospital since it first opened on September 2, 1952.

* * *

Dr. George C. Ham, professor of psychiatry, was guest speaker at the Mountain Top Medical Assembly held at Waynesville, June 22-24.

Dr. Ham, who heads the Department of Psychiatry, delivered two talks. His first presentation was "The Role of the Family Physician in Treatment of the Neurotic Patient" and his second was "The Use and Abuse of Drugs in Management of the Neurotic Patient."

* * *

Dr. Fred. W. Ellis, associate professor of pharmacology, was a guest speaker at a workshop on "Facts About Alcohol" held at Catawba College in Salisbury, June 12-23.

Dr. Ellis spoke on June 13 on "The Physiology and Metabolism of Alcohol." He has long been active in research in this particular field.

The Rowan County ABC Board's Department of Education cooperated with the North Carolina Alcoholic Rehabilitation Program to make this workshop possible. It is one of many such programs that will be held in North Carolina colleges and universities throughout the summer months.

* * *

Miss Ellen Anderson, medical technologist of the Department of Pathology, took office Friday, June 16 as president of the American Society of Medical Technologists.

Miss Anderson assumed her new office at the national convention of the Society held in Seattle, Washington, June 11-16.

Three pschiatrists on the staff of North Carolina Memorial Hospital were cited recently for outstanding theses submitted in connection with their three-year residency training here.

There are Dr. Mario Reyes of Mexico; Dr. John I. Boswell, South Hill, Virginia; and Dr. Eugene Taylor of Moscow, Idaho.

Dr. Reyes took first place and Dr. Boswell was awarded second place for the Anclote Manor Hospital Prize. Dr. Taylor received honorable mention for his thesis.

The medical director of Anclote Manor Hospital of Tarpon Springs, Florida, is Dr. Lorant Forizs, former faculty member of the Department of Psychiatry of the University of North Carolina School of Medicine, who was guest speaker at the presentation exercise.

* * *

Awards have been presented to 22 girls of the Chapel Hill high schools for their participation in the winter Candy-Striper program at North Carolina Memorial Hospital. Certificates, in recognition of 30 hours of voluntary work were awarded to 15 girls, and pins representing 75 hours of work were given to seven of the young ladies.

News Notes from the Duke University Medical Center

A pioneering attack on the problems of the world's children will be launched through an international symposium at Duke University this October.

The five-day meeting will bring together distinguished authorities from the United States and several foreign countries in fields that include education, culture population, economics and health.

Together they will explore the conditions under which the world's children now live and the problems which must be solved if children are to have a better opportunity to become healthy, responsible adults.

Plans for the symposium were announced recently by Duke University President Deryl Hart. Scheduled for October 4-8, the symposium will be entitled "The Commonwealth of Children."

Dr. Hart said that the symposium was conceived as a tribute to Dr. Wilburt C. Davison, who directed establishment of the Duke University School of Medicine and served as dean from 1927 until he relinquished that position a year ago. An internationally known medical educator and pediatrician, Dr. Davidson will retire from the Medical School faculty this summer at the age of 69.

President Hart said the theme "The Commonwealth of Children" was chosen because of its close relationship to Dr. Davison's life and work.

* * *

Duke Hospital has reecived $350,000 from the Hospital and Orphans Section of the Duke Endowment to finance extensive renovation and modernization of facilities.

Hospital Superintendent Charles H. Frenzel said recently that the funds will be used to modernize facilities for patient care and other types of hospital services.

Expressing appreciation for the gift, Frenzel explained that hospitals constantly face the need for updating their facilities to keep pace with advances in medical science and patient care.

* * *

Medical scientists from 12 states and two foreign countries began a month's intensive study of disease-causing fungi on July 3, at the Duke University Medical Center.

The medical mycology course, being given for the fifteenth consecutive year, attracts some 32 physicians and others concerned with fungus diseases.

Dr. Norman F. Conant, James B. Duke Professor and chairman of the Department of Microbiology, is course director.

Emphasis is on the practical aspects of the laboratory as an aid in diagnosing fungus infection.

The course was first given in 1947 in answer to requests from scientists who were acquainted with the Duke Medical Center's Fungus Registry, one of the world's centers for diagnosis and study of fungus diseases.

* * *

Dr. Galen W. Quinn, associate professor of orthodontics at the Duke University Medical Center, has been named dean of the School of Dentistry at Creighton University, Omaha, Nebraska.

Dr. Quinn will assume his new post several months from now after his successor on the Duke Faculty has been appointed.

Dr. Quinn joined the Duke faculty three years ago as the Medical Center's first orthodontist. Earlier, he headed the Department of Pedondontics (children's dentistry) at the University of Tennessee College of Dentistry in Memphis.

His activities at Duke have included work in conjunction with plastic surgeons on cleft palate cases and in conjunction with the surgery department's division of medical speech pathology on mouth conditions that cause defective speech.

Edgecombe-Nash Medical Society

Dr. Floyd W. Denny, newly appointed chairman of the Department of Pediatrics at the University of North Carolina School of Medicine, spoke at the regular meeting of the Edgecombe-Nash Medical Society held in Rocky Mount on July 12. His subject was "Streptococcic Infections and Their Sequelae."

News Notes

Dr. Donald C. Hartzog, Jr., has opened his office for the practice of internal medicine and metabolic diseases in the Professional Building, Cloverdale Avenue, Winston-Salem.

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

The fungus which causes histoplasmosis, a frequently serious lung disease, has been found for the first time in Washington, D. C., the Public Health Service revealed recently.

The fungus, **Histoplasma capsulatum,** was found in soil contaminated by starlings taken from beneath sycamore trees in two of Washington's parks. One of the parks is located at Pennsylvania Avenue between 7th and 8th Streets, N.W. The other is at Massachusetts Avenue and E Street, N.E. Hitherto, many investigators have believed that the fungus grew only in the soil of rural areas and small towns.

The findings, by Dr. Chester W. Emmos, Chief of the Medical Mycology Section of the National Institute of Allergy and Infectious Diseases, are described in the June issue of **Public Health Reports.**

Referring to the site of his latest findings, Dr. Emmons points out in his report that "the park had been periodically and recently cleaned and there was no obvious contamination with bird excreta, although it is known that the soil under the sycamore trees where the collections were made is regularly contaminated with such material. It is obvious that roosting starlings can create a soil environment suitable for the growth of **Histoplasma capsulatum** even though bird excreta do not accumulate and remain on the surface of the soil."

The report emphasized that other scientists also have suggested "an urban type of human exposure" to histoplasmosis and notes that histoplasmosis is already well-documented as "an important medical problem in Washington, D. C., and surrounding areas." In certain Maryland and Virginia communities, the report says up to 83 per cent of the population have shown evidence of past exposure to the disease.

Similar soil isolations of the fungus have been reported from two smaller cities, Milan, Michigan, by Dr. Horace J. Dodge, and Mexico, Missouri, by Dr. Harold J. Lynch.

* * *

Surgeon General Luther L. Terry announced today the appointment of Dr. Richard R. Willey as Deputy Chief of the Division of Research Grants, National Institutes of Health. Dr. Willey was Chief of the Research Grants Branch, Division of General Medical Sciences, National Institutes of Health, from 1958 to 1960. Except for a period of travel and study during the past year, he has been at NIH since 1954, when he became Executive Secretary of the Mental Health Study Section.

American Board of Obstetrics and Gynecology

The next scheduled examination, (Part I), written, will be held in various cities of the United States, Canada, and military centers outside the Continental United States on Friday, January 5, 1962.

Case Reports are no longer required by this Board to complete the Part 1 Examination.

In lieu thereof, all applicants and candidates for examination are required to submit a **duplicate certified typewritten list** of patients dismissed from each hospital during the preceding 12 months. This applies to new applicants, reopened applications, and candidates requesting re-examination in Part I or Part II examination.

Lists of obstetrical and gynecological patients are to be made separately and must conform in all details to the sample format furnished upon request by the office of the executive secretary and treasurer.

Candidates are no longer required to bring a duplicate list of admissions to the Part II examination.

Current Bulletins may be obtained by writing to: Robert L. Faulkner, M.D., Executive Secretary and Treasurer, 2105 Adelbert Road, Cleveland 6, Ohio.

Society of Nuclear Medicine

The Society of Nuclear Medicine recently concluded its eighth annual meeting in Pittsburgh, Pennsylvania.

Prof. George C. de Hevesy of the University of Stockholm, Sweden, winner of the 1943 Nobel Prize in Chemistry and the second Atoms for Peace Award in 1959, presented the second of the Annual Lecture Series— a series of lectures dedicated to pioneers in nuclear medicine—in honor of Marie and Pierre Curie, and Henri Becquerel.

The following officers were elected: president—Lindon Seed, M.D., Chicago; president-elect—J. R. Maxfield, Jr., M.D., Dallas; vice president—Herbert C. Allen, Jr., M.D., Houston; vice president-elect—Joseph Sternberg, M.D., Montreal, Canada; secretary—Robert W. Lackey, M.D., Denver; treasurer—William H. Beierwaltes, M.D., Ann Arbor, Michigan; historian—Asa Seeds, M.D., Vancouver, Washington.

The ninth annual meeting of the society will be held at the Baker Hotel, Dallas, Texas, June 27-30, 1962.

For further information, address all inquiries to the Administrator, Society of Nuclear Medicine, 430 North Micigan Avenue, Chicago 11, Illinois.

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American College of Physicians

Included in the autumn schedule of postgraduate courses sponsored by the American College of Physicians in 1961 is a course on "The Physiologic Basis of Internal Medicine, " which will be held at the Duke University Medical Center in Durham, October 9-13.

Directors of the course will be Drs. Eugene A. Stead, Jr., and Elbert L. Persons.

This course will deal with recent progress in the understanding, diagnosis, and treatment of disease in the major areas of internal medicine. Emphasis will be on the physiologic mechanisms underlying the disease process.

All courses have been arranged through the cooperation of the directors and the institutions at which the course will be given. Tuition fees are $60.00 for members and $80.00 for non-members. Registration forms and requests for information should be directed to Edward C. Rosenow, Jr., M.D. Executive Director, American College of Physicians, 4200 Pine Street, Philadelphia 4, Pennsylvania.

American Urological Association

The American Urological Association offers an annual award of $1000 (first prize of $500, second prize $300, and third prize $200) for essays on the result of some clinical or laboratory research in urology. Competition is limited to urologists who have been graduated not more than ten years, and to hospital interns and residents doing clinical or laboratory research work in urology. Animal research is not necessary.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Bellevue-Stratford Hotel, Philadelphia, Pennsylvania, May 14-17, 1962.

For full particulars write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore 1, Maryland. Essays must be in his hands before November 15, 1961.

College of Medical Evangelists

The last student has been graduated from the College of Medical Evangelists. The 56 year old medical education center with campuses here and in Los Angeles has conferred its last degree.

As flashbulbs popped, Mrs. Glennis Eugena Burke, mother of two and a specialist in a field of nursing administration, was informed that she was officially the last graduate of her alma mater, which assumed the name of Loma Linda University on July 1. All future graduates will receive diplomas bearing the University name.

First classes in the new University begin in September. Two liberal arts colleges—La Sierra College in Arlington and Pacfic Union College in Angwin—are associating with the Southern California school in the University organization, according to an official announcement from the Seventh-day Adventist educational institution here.

National Fund for Medical Education

Contributions from industry to the National Fund for Medical Education in 1960 reached a new high of $2.418,221, S. Sloan Colt, president of the National Fund, announced recently.

These figures were contained in the 1960 Annual Report of the Fund. This sum represents an increase of 8.8 percent over money contributed in 1959. Aided by a matching grant of $919,000 from the Ford Foundation, the Fund was enabled to make awards to the nation's 85 medical schools of $3,138,460. This is an increase of $100,000 over grants made in 1959.

More than 1,900 corporations contributed to the fund in gifts ranging from $100,000 to $5.00. In addition, some $23,219 was received from individuals for the Fund's Teaching Budget Program.

American Association of Industrial Nurses

A classified advertising section will be carried in the **Journal** of the American Association of Industrial Nurses as a service to employers seeking industrial nurses and industrial nurses seeking positions in this field. The announcement was made by Ella Casey, R.N., executive director of the A.A.I.N., which is the national organization of the industrial nursing specialty.

The A.A.I.N. **Journal**, the only nationally circulated publication devoted exclusively to industrial nursing, is published monthly and distributed to members of the Association, physicians, management representatives, and others with an interest in occupational health. Rates and information concerning the placement of classified advertisements may be obtained from Advertising Representative, American Association of Industrial Nurses, 170 East 61st St., New York 21, New York.

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Veterans Administration

With a nationwide shortage of professional nurses, is their working time being wasted on clerical and other chores that could be done by non-nursing personnel?

To its 170 hospitals, the Veterans Administration has sent "fresh-off-the-press" copies of a new VA publication that could revise currently-accepted practices in hospital nursing, to permit nurses to spend more time in direct care of patients.

Entitled "A Guide For Studying The Utilization Of Nursing Service Personnel In VA Hospitals," the volume was developed with the aid of Columbia University, to enable each VA hospital to make a valid study of how it uses—or possibly misuses—its nursing personnel.

Miss Cecilia H. Hauge, the VA director of nursing in Washington, D. C., said she hopes the Guide will be of value to hospitals outside the VA, as well as to the VA's medical installations. Copies will be available from the Superintendent of Documents, U. S. Government Printing Office, Washington, D. C.

* * *

Appointment of Dr. Robert E. Riederer of Olathe, Kansas, as chief of the professional training division in the Education Service of the Veterans Administration Department of Medicine and Surgery, Washington, D. C., was announced recently.

Dr. Riederer has been in private practice in Olathe since 1946.

The World Medical Association

The Fifteenth General Assembly of the World Medical Association will convene at the Copacabana Palace Hotel, Rio de Janeiro, Brazil, September 15-20, 1961.

Immediately preceding the Assembly from September 11 to 15 the Brazil Medical Association will hold a symposium on Arborvirus.

Conclusions of this symposium will be presented in the scientific session of the General Assembly on Saturday, September 16, 1961.

As a special feature the Instituto Britantan will provide demonstrations of the techniques of extracting venom from snakes.

Special topics on the program of the General Assembly will include consideration of Increased Cost of Medical Care under Social Security Schemes, Draft Code of Human Experimentation, and World Problems of Medical Education.

* * *

On July 3, 1961 Dr. Harry S. Gear of Johannesburg, South Africa succeeded Dr. Louis H. Bauer of New York as Secretary of The World Medical Association.

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JOHN D. PATTON, M.D. Clinical Director

The Month in Washington

The American Medical Association opposed three major provisions of a bill (S. 1552) that would greatly increase the powers of the federal government in regulation of the ethical drug industry.

These three provisions would turn over to the Department of Health, Education and Welfare and the Food and Drug Administration the responsibility for (1) relaying of drug information to physicians, (2) selecting the names of new drugs, and (3) deciding whether a drug is of value in treating human ills.

The A.M.A. did not take a position on the bill as a whole because certain of its provisions, "such as the Sherman Act and patent law amendments, are outside our area of competence."

Dr. Hugh H. Hussey, Jr., chairman of the A.M.A.'s Board of Trustees and dean of Georgetown University (Washington, D. C.) School of Medicine, was the chief A.M.A. witness at the opening of hearings on the legislation before the Senate Antitrust and Monopoly Subcommittee headed by Sen. Estes Kefauver (D., Tenn.). Dr. Hussey was accompanied by Dr. Ernest B. Howard, assistant executive vice president of A.M.A. and C. Joseph Stetler, A.M.A.'s general counsel.

With Congress trying for adjournment by about September 1 and much "must" legislation still to be acted upon, it appeared highly unlikely that Congress would complete action on the drug legislation this year.

Dr. Hussey reviewed for the subcommittee A.M.A.'s 70-year record of leadership in endorsing legislation designed to insure the purity of drugs and food. The A.M.A. carried on intensive legislative efforts in the field and "is generally credited with being one of the major forces that brought the first Pure Food and Drug Act into being" in 1906, Dr. Hussey said.

Dr. Hussey cited these A.M.A. aims that "we, as physicians, are desirous of achieving:

From the Washington Office of the American Medical Association.

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"—We want all physicians to be well-trained and fully informed on all aspects of the practice of medicine.

"—We want this body of knowledge and reseroir of skills to include a high degree of competence in the selection and proper use of drugs.

"—We want a continuing and expanding flow of useful drug products placed at the disposal of these physicians."

Dr. Hussey pointed out that the A.M.A. already conducts an intensive program of informing physicians about new drugs and that this program is now in process of being greatly stepped up.

"The medical profession believes that the education of physicians is the responsibility and preogative of the profession itself," he said.

Assigning responsibility for selecting names of new drugs to the federal government would merely be duplication of the program of drug nomenclature which has been operated for many years by the A.M.A. and the pharmaceutical industry, Dr. Hussey declared. This program also has recently been refined and improved, and will continue to meet the need for an orderly system for selecting names for new drugs.

In the final analysis, it is the physician and the pharmacist who must know the non-proprietary names of drugs, he said. These two professions now direct this naming process, and "we do not believe the responsibility for designating and revising names should be assigned to a governmental agency," he said.

Regarding determination of the efficacy of a new drug, Dr. Hussey said:

"We believe that only the physician has the knowledge, ability and responsibility to make a decision as to what drug is best for a particular patient. He should not be deprived of the use of drugs that he believes are medically indicated for his patient by a governmental ruling or decision.

"Physicians seek to treat the medical problems of *individual patients*. A physician does not treat 10 cases of hypertension; he treats 10 individual patients, each of whom has a medical problem he has diagnosed as hypertension. He may find that the same dosage of the same form of the same drug will be efficacious in each and all of his ten patients.

"Or he may find that one or more of them need different dosages, or different forms of this same drug. He may, indeed, find that one, two or three of them are allergic to the non-active ingredients used in this brand of the drug, and that a different brand, with other non-active ingredients, is the proper answer.

"Thus, in one patient, a specific dosage of a specific drug might be said to be efficacious, while in another it would be described as totally ineffective.

"A physician can be told many things about a drug, including its chemistry, its mode of action and, to some extent, its toxic properties. But he must judge its efficacy."

Metrecal Diet Helps Diabetics Lose Weight

Overweight diabetic patients showed marked improvement and, in many cases, were able to reduce or totally eliminate their requirements for insulin after reducing their weight with Metrecal, according to a report presented at the American Medical Association's annual convention recently. The findings were described in a report on a clinical study of 101 adult diabetic patients who had a history of being "consistently and uniformly unsuccessful in repeated attempts at weight reduction."

Clinical investigators submitting the report were Seymour L. Halpern, M.D., and Anafred N. Halpern, M.D., of the New York Medical College, Metropolitan Medical Center, New York City. Dr. Halpern is president of the Food and Nutrition Council of Greater New York and chairman of the Subcommittee on Nutrition, Medical Society of the County of New York.

The point was made that while Metrecal was very successful in achieving weight loss, the improvement in diabetic condition occurred because the weight was lost, not because of the means used to lose it.

In addition, the clinicians focused attention on the importance of diabetics undertaking weight control only under the direct supervision of a physician when they cautioned doctors attending the A.M.A. convention that adjustments must be made in the patient's insulin (or related drug) therapy concurrently with the introduction of a change in the diet in order to avoid complications in the delicately balanced metabolism of a diabetic patient.

In Memoriam

Arthur Lee Daughtridge, M.D.

Suddenly and without warning, on the evening of May 10, 1961, our friend and colleague, Dr. Arthur Lee Daughtridge, died unexpectedly at the Rocky Mount Sanitarium of an acute coronary thrombosis.

Dr. Daughtridge, a native of Rocky Mount, was born October 21, 1900. He was the son of Margaret Elizabeth Joyner and the late Robert Lee Daughtridge. He came from a large and prominent family in this community, both sides of his family having been established here prior to the Revolutionary War. Dr. Daughtridge spent his life in Rocky Mount, where he was a graduate of the Rocky Mount High School. He early joined his life to that of the church and was a member of the First Baptist Congregation in Rocky Mount. He was a charter member of the first Boy Scout Troop which was sponsored in Rocky Mount, Troop No. 1.

Dr. Daughtridge obtained his education at the University of North Carolina, obtaining his B. S. Degree in medicine at the age of 22. Having received his two years in medicine there, he entered the University of Maryland, where he graduated in 1924. He spent the year 1924 at City Hospital, Batimore, Maryland, and then returned to Rocky Mount, where he became affiliated with the late Dr. L. W. Kornegay at the Rocky Mount Sanitarium, Inc.

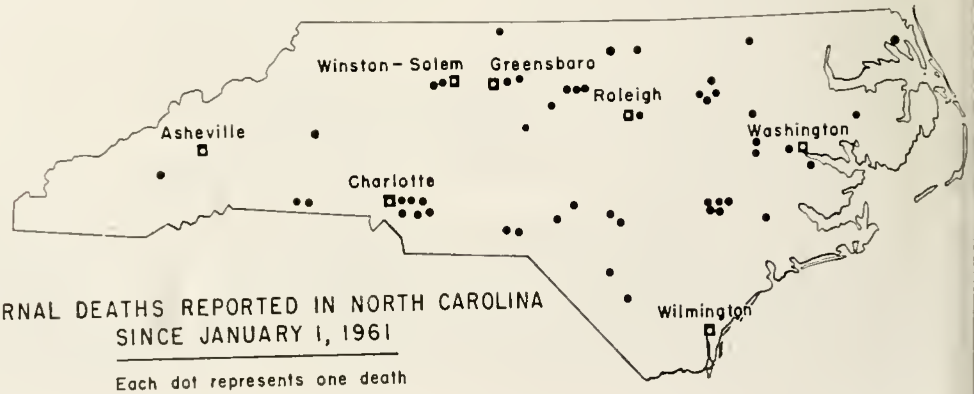
Dr. Daughtridge had charge of the medical department of the hospital when he first entered the Sanitarium, but his main interest was in roentgenology, in which he finally specialized after receiving postgraduate instruction at Polyclinic in New York.

Dr. Daughtridge was prominent in the professional and social life in this town. He held many offices of importance in organizations in this area. He was president of the following medical organizations: The Edgecombe-Nash County Medical Society, the Fourth District Medical Society, the North Carolina Radiological Society, and the North Carolina Hospital Association. He was the vice president of the Medical Society of the State of North Carolina. He was also president of the Tri-State Hospital Association and a member of the Joint Stabilization Board of the North Carolina Nurses Association. In recent years he served as chairman of Public Relations of the county medical society. He was also a member of the Executive Committee of the Medical Society of the State of North Carolina.

Dr. Daughtridge gave of his time and energies unselfishly to many community causes. He was especially interested in the Rocky Mount Tuberculosis Association, of which he was president at the time of his death. He was also interested in the Cancer and Heart Associations. An active Kiwanian, he was Lieutenant Governor of the Sixth Carolina District, and headed the work of the Boys and Girls Committee. In addition, he served as president of the Children's Museum. Dr. Daughtridge was especially interested in all the activities related to the youth of the city.

Dr. Daughtridge was greatly beloved by the people of this community. He loved people, and people loved him. Therefore, **Be it resolved** that the Edgecombe-Nash County Medical Society and the Citizens of North Carolina have lost not only an upstanding member of the medical profession, but an outstanding citizen generally. **Be it further resolved** that a copy of this Resolution be attached to the permanent minutes of this meeting, a copy be sent to Dr. Daughtridges' family, and a copy to the State Medical Society.

D. L. Knowles, M.D., Chairman
R. M. Whitley, M.D.
R. D. Kornegay, M.D.



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Also: Transactions and Roster of the Auxiliary

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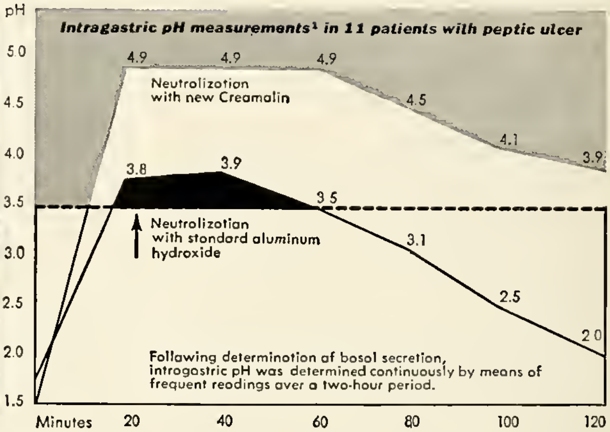

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NORTH CAROLINA MEDICAL JOURNAL

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Some Recent Clinical and Experimental Advances Relative to Diseases of the Biliary Tract and Pancreas

WILLIAM W. SHINGLETON, M.D.
DURHAM

Diseases involving the biliary tract and pancreas have been the subject of many nteresting and informative studies within recent years. Better understanding has come rom both animal experiments and clinical observations. Many problems, however, remain unsolved, and much work is still in progress. In the following discussion, some of the more significant laboratory and clinical studies have been chosen for brief discussion, with the realization that they constitute only a small fraction of the total vork going on in this area.

Pancreatic Diseases

Progress in the study of pancreatic disease has been slow but steady. Improvement in treatment has come mainly from tudies which have better defined the normal and abnormal anatomy and function f this gland. Especially noteworthy is consideration of the possible role of the pancreas in extrapancreatic diseases—for instance, peptic ulcer and disease of the biliary ract. Re-emphasis of a relationship between ancreatic function and peptic ulcer has ollowed the description by Zollinger and Ellison[1] of a relationship between non-beta islet cell tumor of the pancreas and peptic ulcer. Although initally it was thought that astric hypersecretion in this instance was ue to excessive glucagon secretion by these umors, studies failed to confirm this theory. ecently, however, a substance, still undentified, has been extracted from such tumors which apparently does produce gastric hypersecretion[2].

There is also evidence to suggest that the pancreas may likewise play a role in protection against peptic ulcer. According to Longmire[3], Dragstedt, in 1922, reported a very high incidence of peptic ulcer of the duodenum in dogs after total pancreatectomy or following creation of a total pancreatic fistula. More recently, several groups of investigators have shown a significant increase in acid secretion in a Heidenhain pouch in dogs following interference with pancreatic function—namely, after pancreatectomy, ligation of the pancreatic ducts, or alloxan diabetes[4]. Workers are now trying to determine if a substance is elaborated by the pancreas which plays a role in control of gastric secretion.

Studies on the etiology of pancreatitis continue. There are several recent reports on the occurrence of acute pancreatitis following operative procedures in the upper part of the abdomen[5]. Several points emerge from these reports. When this complication does arise, it is often fatal. In one series of 75 patients with postoperative pancreatitis, the mortality was 77 per cent. The two most common operative procedures leading to this complication were subtotal gastric resection and exploration of the common bile duct.

An interesting new approach to the etiology of chronic pancreatitis is the possibility that this condition may be an autoimmune reaction, much as chronic thyroiditis is now regarded as a hypersensitivity phenomenon[6]. In these studies, animals have been immunized against homogenates

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From the Department of Surgery, Duke University edical Center, Durham, North Carolina.

Read before the Section on Surgery, Medical Society of e State of North Carolina, Asheville, May 6, 1961.

of their own pancreas, with the development of antibodies to this antigen. In other studies of patients with pancreatic disease, nuclear fractions of the human pancreas were used as antigens; and in one study, significant antibody titers were found in the serum of 32 of 36 patients with known chronic pancreatitis, 9 of 12 patients with proven carcinoma of the head of the pancreas, and 6 of 7 patients with cystic fibrosis[3]. Control studies, using blood from a blood bank, were all negative. If further research confirms these findings, the implications are extremely important from the standpoint of diagnosis and treatment.

Another area of study with implications concerning the treatment of chronic pancreatitis is renewed interest in the anatomy of the pancreatic ductal system. For some years argument has been going on as to whether intrapancreatic ductal obstruction is a significant etiologic factor in relapsing pancreatitis. Warren[5], who has had considerable experience with operations on the pancreas, believes that ductal obstruction is a common finding in chronic pancreatitis. This opinion is based on pancreaticograms, exploration of the pancreatic duct at operation, and pathologic examination of resected portions of the pancreas. Birnstingl[7], in autopsy studies of the pancreatic duct in 150 subjects who had had no known pancreatic disease, found the main duct to vary in caliber between 2 and 9 mm., with an average volume of 2 mm. of dye required to inject the ductal system. Berman[8] injected 143 pancreatic ducts with vinyl acetate or latex at autopsy, and in 82 per cent of the subjects the duct of Wirsung joined the common bile duct 2 to 3 mm. proximal to the ampulla of Vater. In 6 per cent of the cases the duct of Wirsung emptied into the common bile duct 5 to 15 mm. proximal to the ampulla; in 6 per cent there were independent openings of the two ducts at the ampulla, and in 6 per cent no duct of Wirsung was present. These injection studies showed that there is some variation in the size and tortuosity of the pancreatic duct between individuals. However, the collected experience of several surgeons now indicates that the incidence of stenosis or obstruction of the main pancreatic duct in chronic relapsing pancreatitis is quite high. This has been our experience in recent operations for this disease.

Disease of the Liver

The liver continues to be an organ of great interest to the research worker. The livers of several experimental animals, including the rat, the dog, and the calf, or being perfused after removal from the animal are found to survive and continue to function for variable periods. The clinical application of this finding is the possibility of using an extracorporeal liver to sustain patients with severe liver damage for temporary periods until their own liver recovers. Also, the liver, along with many other organs, is being studied in regard to homotransplantation[10]. Studies in animals thus far suggest that the type of rejection phenomenon demonstrated with regard to the liver is similar to that in renal homotransplantation—namely, that the organ will survive and function in the host animal for five to seven days, and then be rejected by the host. Many efforts are being made to alter the host reaction so that these organs will survive and function for longer periods of time. When and if this problem is solved, an entirely new field of surgery will unfold.

Of growing importance in the study o liver disease is catheterization of the hepati vein, first described in 1944[11]. This technique has permitted measurement of hepatic vein and sinusidal pressures, estimation of hepatic blood flow, studies of uptake and turnover of drugs and metabolites, and retrograde visualization of the venous network of the liver by means of contrast media. These data, correlated with clinical and other findings, have increased the diagnostic and therapeutic perspective in patients with liver disorders, and have provided much basic information on hepatic function in health and disease.

The problem of cirrhosis following infectious hepatitis has interested students o liver disease for some time. In a recen article, Ratnoff[12] stated that while the incidence of this sequela is still not known, definitely occurs, and that two varieties ar seen. One is referred to as postnecrotic cirrhosis, in which the pathologic process consists of necrosis of liver cells with regeneration of hepatic tissue and the formation of large bands of connective tissue coursing through the liver. In some parts of the liver, the normal hepatic architecture is preserved. This is in contrast to Laennec's cirrhosis, where the liver typically shows a diffuse fibrosis involving all the lobules, with disruption of the normal relationship within the lobules. It is so diffuse that every microscopic field appears to be involved. Approximately 5 to 10 per cent of patients with Laennec's cirrhosis give a history suggesting infectious hepatitis which usually has occurred several years prior to the onset of clinical cirrhosis. The factors determining whether cirrhosis develops in a patient who has had hepatitis are not known. There seems to be no correlation between the severity of the hepatitis and the eventual development of cirrhosis. Cirrhosis following hepatitis may be associated with bleeding esophageal varices, which may require operative management.

Resection of a lobe of the liver for benign and malignant conditions has been reported with increasing frequency in recent years[13]. The major problem involved in resection of the liver is that of uncontrolled hemorrhage and infection. Based on a better understanding of the anatomy of the organ, techniques for safely removing either lobe of the liver have been developed and with the advent of wide-spectrum antibiotics have made possible the satisfactory control of infection.

Inasmuch as the liver is responsible for so many biochemical processes in body metabolism, there has been considerable fear regarding the maintenance of normal metabolism following resection of large portions of the liver. For example, resection of the entire right lobe involves removal of 80 per cent of the total liver substance. Pack[14], who has had considerable experience in liver resection, reported recently on the metabolic effects of hepatic lobectomy in 23 patients. The most striking is the liver's regenerative capacity. The rate of regeneration is inversely proportional to the size of the animal. Only two to three weeks are required for regeneration in the rat; in the human, approximately one year is required. The immediate effects of lobectomy of the liver in man reflect both parenchymatous and obstructive changes. There is a moderate rise in the serum bilirubin and alkaline phosphatase, an increase in the cephalin flocculation and thymol turbidity values, increased retention of bromsulphalein, decrease in the prothrombin time, and a reduction in the serum albumin. All these effects, are transient however, and usually by the fifth day all values except the serum albumin are back to near normal. Serum albumin values return to normal in about three weeks. No marked changes in plasma electrolytes occurred.

Although the indications for hepatic lobectomy are not yet well defined, the procedure has been used for localized primary or metastatic tumors confined to one lobe of the liver, for hemangiomas, carcinoma of the gallbladder, and traumatic injuries. A five-year cure for carcinoma of the gallbladder by right hepatic lobectomy was recently reported[15]. Wilson[16] has recently described resection of the right lobe of the liver by use of general body hypothermia and cross-clamping the aorta and vena cava above the diaphragm.

Biliary Disease

The problem of biliary atresia remains an enigma for the surgeon. Krovetz[17] recently summarized the experience in the Department of Pediatrics of the University of Minnesota and reviewed the literature on this subject. The study involved 137 cases of congenital biliary atresia in patients born after January 1, 1945. Of these cases, 15 were of the intrahepatic variety, and 76 lacked extrahepatic ducts amenable to surgery. The review indicated that in only 19 per cent of the cases studied were there ducts theoretically suitable for operation. He stated that the number of cases actually cured by operation was discouragingly small, and estimated that the cure rate to be expected in patients with this condition is about 8 per cent. An interesting aspect of the study was that 14 patients thought to have an inoperable condition and a hopeless prognosis subsequently recovered from jaundice.

Theoretically, the operation of choice would be the one which most nearly duplicates normal anatomic biliary flow. However, from the data reviewed there did not seem to be any obvious choice in terms of operative success. The author was unable to find a single surgical success following hepatoenterostomy, internal or external drainage of bile. He suggests that, since operative diagnosis is not infallible, the procedure of choice, if no correctable atresia can be found, would seem to be not to attempt these procedures at initial operation.

Opinion varies as to optimal timing of surgical intervention in these patients. Although fibrotic change progresses with age, there are reports of cases marked by severe fibrosis early in life, but little or none with increasing age. The author concludes from analysis of the cases that the patient should be observed for at least four weeks or until the age of 3 months before operation.

There are a variety of causes, such as retained cystic duct stump or common duct stones, for the continuation of symptoms following cholecystectomy. A relatively common one appears to be stenosis of the sphincter of Oddi. Variously called sphincteritis, fibrosis of the sphincter, and stenosis of the sphincter, this condition may be encountered while exploring the duct at operation for chronic relapsing pancreatitis, or during exploration of the common duct in conjunction with stones in the gallbladder or common duct. However, it apparently is encountered more often in patients who continue to have symptoms following cholecystectomy for chronic cholecystitis and cholelithiasis. Cattell[18] is credited with describing the condition, and his experience with it has been large. In a recent report of 100 cases, 81 of the patients had had a previous cholecystectomy without relief of their original symptoms. Forty of these 81 had had a choledochostomy at the time of the cholecystectomy. Forty-six of the 100 patients were jaundiced or gave a history of jaundice. He feels that the most useful laboratory aid in diagnosis is the intravenous cholangiogram. In this study, both the size of the duct and its rate of emptying are important. Studies have shown that the average common duct varies in diameter from 8 to 15 mm. A duct larger than 15 mm. in diameter is considered abnormal. Retention of dye in the duct longer than two hours suggests obstruction. The diagnosis is established when, at operation, one is unable to pass at least a 3 mm. Bakes' dilator through the ampulla of Vater. A duodenotomy is then performed, and the stenotic sphincter directly visualized and cut. This procedure has proved effective in relieving symptoms.

The final subject that I would like to mention briefly relates to a new approach to visualization of the extrahepatic biliary system by percutaneous transhepatic cholangiography. This procedure was first described in 1952, by Carter[19], but has been used infrequently since that time. Several reports have come from South American countries[20]. A needle is introduced through the anterior abdominal wall in the right upper quadrant and directed toward the hilum of the liver. At periodic intervals aspiration is carried out and when an aspirate of bile is obtained, radioopaque dye is injected and an x-ray picture made. The object is to introduce the needle into one of the intrahepatic radicles of the biliary tree, since introduction of the needle into a gallbladder or common bile duct is fraught with danger from leakage of bile into the peritoneal cavity. Candidates for the procedure are patients who have jaundice and, more specifically, those in whom intrahepatic and extrahepatic bile duct obstruction cannot otherwise be differentiated.

Summary

Several areas of research relative to diseases of the pancreas and biliary tract have been discussed briefly. Definite progress toward a better understanding of these diseases is being made. Current research is pointing toward an expanding area of surgical therapy in diseases involving the pancreas, liver, and biliary system.

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Recent Clinical and Experimental Advantages in Surgery of the Colon & Rectum

COLIN G. THOMAS, JR., M.D.
CHAPEL HILL

The past decade has witnessed a number of changes in the surgical management of diseases of the large intestine, which for the purpose of this discussion are grouped euphemistically under the broad category of "progress." Progress in medicine connotes improvement in diagnostic skills and the associated recognition of well established or new disease entities. In addition, the term implies more successful methods of therapy, which are hopefully ascribed to a better understanding of the mechanisms of disease but actually are more frequently empiric in origin. For these reasons, this presentation is more in keeping with a "report in progress." Only with a better understanding of pathologic mechanisms and the use of the "retrospectroscope" will we be able to evaluate the validity of today's "advances."

In the time allotted it is obviously impossible to review all aspects of surgery of the large bowel. I should like, therefore, to discuss two primary problems—namely, diverticular disease of the colon, and the role of chemotherapy as an adjuvant to the surgical treatment of cancer. In addition, there are a few diagnostic and therapeutic techniques which warrant comment.

Diverticular Disease of the Colon

Diverticulosis and diverticultitis have long been recognized as conditions associated with advancing age. In patients past the age of 60, approximately one third will show radiologic evidence of diverticulosis, and less than one third of these have symptoms. It is important, therefore, not only that the vagaries of this disease be well known, but that the role of surgical treatment be well defined.

Until recently the prevailing attitude toward diverticulosis was expressed in the

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1941 edition of Cecil's Textbook of Medicine[1]. "In the great majority of cases diverticula produce few or no symptoms and are accidentally found. Perforation occurs very rarely; inflammation is not uncommon but it usually subsides with treatment; abscess occasionally develops and rarely chronic inflammation with connective tissue formation produces sufficient obstruction to cause operative treatment."

The ability to control bacterial growth within the gastrointestinal tract, concomitant improvement in anesthesia, blood and fluid replacement, and over-all postoperative care, as well as certain technical considerations— these have made resection and anastomosis of the colon a relatively safe procedure. This fact, together with a more critical appraisal of the morbidity and mortality associated with the "non-surgical" treatment of diverticulitis, as well as the morbidity associated with the surgical treatment of complications, has broadened the indications for surgical treatment of diverticular diseases of the colon. Many reports of the "successful" results of such therapy are now accumulating.

There would seem to be little debate regarding the need for surgical treatment of the complications of diverticulitis—namely, (1) partial or incomplete obstruction that one is unable to differentiate from a neoplastic process; (2) perforation with peritonitis or abscess formation; (3) fistula formation: (4) recurrent or massive hemorrhage, and (5) recurrent attacks of diverticulitis. Considerably more judgment must be exercised, however, as to advisability of "prophylactic resection" in patients with diverticulitis in order to avoid these complications. Obviously, the removal of a localized segment of colon with normal bowel for anastomosis is quite a different problem from that presented by more extensive disease with pericolitis extending from the rectosigmoid to a point high in the descending colon. Furthermore, recognition of the extent of the disease by radiologic and clinical appraisal is notoriously unreliable.

In evaluating the indications for "prophylactic" surgery in patients with diverticulitis, or for operations, as one author has expressed it, to "alleviate symptoms and to avert complications which seem predictable in the near future," one should bear in mind not only the natural history of the disease, but also the results, morbidity, and mortality of surgical treatment. Unfortunately, few reports have emphasized the natural history of *diverticulosis* in a specific manner. Perhaps the most detailed is that given by Boles[2], in recording his experience with 294 patients having a mean age of 55 for an average follow-up period of 15 years. In 70 per cent of these patients there was progressive involvement of the colon by diverticulosis. Forty per cent of the entire group developed the following complications: (1) diverticulitis—25 per cent; (b) hemorrhage — 5 per cent; (c) fistula—5 per cent; (d) obstruction—5 per cent.

Two thirds of the patients with obstruction or fistula also had clinical manifestations of diverticulitis. Despite this relatively high incidence of diverticulitis, more than one half of the patients had only one attack. Multiple attacks were associated with twice as many other complications. The incidence of other complications with active diverticulitis was 38.4 per cent as compared with 8.1 per cent for patients with diverticulosis alone. Of the 73 patients with the complications of diverticulitis, 16 (22 per cent) required surgery for either obstruction, perforation, fistula, or recurrent diverticulitis.

In addition to being familiar with the natural history of the disease, one must appraise the results of surgical treatment. There are almost no reports on the long-term results of extirpative surgery. Although the disease primarily involves the sigmoid colon, it is progressive and can only be completely abolished by total colectomy Subsequent involvement of other portions of the colon are not unlikely, and the chief value of surgical treatment would seem to be quantitative, in reducing the likelihood of inflammatory disease.

Despite the many reports of the success of surgical management, careful scrutiny reveals the morbidity and mortality to vary from 1.5 per cent to as high as 25 per cent in some series (table 1) [3-11]. Even in those

Table 1

Surgical Treatment of Diverticulitis

Author		Location	Patients		Mortality
Bacon	'61	Temple	135		1.5%
Brown	'60		64 194	conservative Rx	39.0% 6.0%
Browne	'59	Tulane	37		0.0%
Colcock	'58	Lahey Clinic	102		1.5%
Hinton	'59	New York	82	40 elective 42 urgent	7.3%
Kratzer	'60	Allentown	24	18 elective 6 urgent	0.0%
Quinn	'60	Charity	20		25.0%
		Salt Lake City	51		5.9%
Welch	'58	Boston	218		2.7%

Table 2

Surgical Treatment of Diverticulitis

Author		Location	Patients		Mortality	Morbidity
Berry	'61	Michigan	53		1.9%	29%
Boyden	'60	Portland	46		8.6%	65%
Jew	'60	Pittsburgh	40		2.5%	40%
Mahon	'59	Dallas	10	(1 stage)	0 %	30%
O'Brien	'59	Ontario	64		9.5%	31%
Schlicke	'59		70	54 elective 16 urgent	2.8%	28%
Smithwick	'60	Boston	30		0 %	26%
Waugh	'57	Mayo	93	(1 stage)	1.4%	17%
			220	(multiple stages)	4.1%	

series with a low mortality, the morbidity has ranged from 17 to 40 per cent (table 2)[12-19]. For these reason the approach to diverticulitis should be basically conservative, and I would agree with Berry[12] as to the indications for operation:

> The emergent surgical considerations are those of **acute obstruction, perforation into the free peritoneal cavity, perforation with extensive pelvic abscess,** and **exsanguinating hemorrhage.** In the absence of these findings, and in view of the fact that 80 per cent of patients can be treated conservatively, the decision for operative intervention may be as follows:
>
> 1. Inability to differentiate an inflammatory mass from neoplasm (25%).
> 2. Repreated attacks of diverticulitis necessitating hospitalization.
> 3. Failure of resolution of an acute process with adequate conservative treatment.
> 4. Progressive symptoms of urinary tract involvement or of demonstration of a fistula.
> 5. Other internal or external fistulae.
> 6. Massive exsanguinating hemorrhage.

In elective surgery a one-stage resection and anastomosis should be possible in most patients with appropriate preparation. The use of an obstructive type of resection for emergent procedures and for conditions not suitable for primary resection and anastomosis should make it possible to restore gastrointestinal continuity in many patients by a two-stage procedure. This approach should lower the morbidity as well as decrease the period of hospitalization.

Although diverticulitis of the right colon is a rare disease, familiarity with its manifestations is essential for its early recognition. The lesion is more likely to occur one or two decades earlier in life than does left-sided diverticulitis, with which it is frequently associated. The diverticulum is anatomically false, and may vary in size from a few millimeters to a size suggestive of reduplication of the colon. Inflammatory changes are usually on an obstructive basis, and, as on the left side, perforation is rare. The presenting symptoms are usually mid- or right lower abdominal discomfort in association with a mass or inflammatory changes in the right lower quadrant. The disease commonly masquerades as acute appendicitis, although the course is usually more gradual in onset and development. A mass may be palpable in one third of the patients. The differential diagnosis includes appendicular abscess, carcinoma of the colon with perforation, regional enteritis, and acute cholecystitis.

In patients coming to operation the procedure will depend on the findings—that is, (1) drainage of an abscess, (2) local resection, or (3) right colectomy with an ileocolostomy. In our experience the diagnosis frequently has not been made until the time of operation or examination of surgical specimen.

Prophylactic Measures in Surgery of Carcinoma of the Large Bowel

During the past five years a number of investigators have emphasized the role of chemoprophylaxis against the spread of cancer during the extirpation of neoplasms involving the gastrointestinal tract[20-22]. These considerations have been based primarily upon the following: (1) the demonstration of neoplastic cells in the venous blood-draining tumor sites; (2) demonstration of neoplastic cells in peritoneal fluid; (3) the relatively high incidence of recurrence involving the line of anastomosis, particularly in low resections; and (4) the low but definite incidence of wound recurrences without other evidence of metastatic disease.

In an effort to minimize recurrence due to local spread at the time of operation, the following measures have been advised: (1) ligation of the bowel proximal and distal to the lesion prior to mobilization and resection; (2) ligation of the venous and arterial blood supply early in the operative procedure; (3) a protective covering for the lesion during operation; and (4) the use of chemotherapy — intraluminally, intraperitoneally, and systemically administered. The first three measures have not been employed long enough to be evaluated adequately. The spread of neoplastic disease both by direct dissemination and by lymph and blood stream is not an isolated phenomenon, but is essentially continuous. It is likely that the vast majority of cells do not survive. These measures, then, would be employed during a relatively infinitesimal portion of the life of the tumor and its potential spread.

So far as local chemotherapy is concerned, recommendations have been based primarily on the findings in experiments on animals. In the rabbit, after the intraluminal inoculation of tumor cells, excision of a segment of colon using an open technique was followed by an 83 per cent incidence of local as well as peritoneal recurrence[23]. Of almost equal interest was the observation that recurrence was highest when the colon had been prepared by the use of antibiotics The only significant clinical studies to date are those of Golligher and colleagues (cited by Keynes[20]), who for a number of years have used mercuric bichloride (1:500) as an agent for cleansing the colon, as well as for lavage of the perineal wound. In thei experience, the incidence of local recurrence at the line of anastomosis in anterior resections was reduced from 19 to 2 per cent in association with the use of mercuric bichloride.

Recently a number of observers have advocated the use of Chlorpactin-XCB both a a cleansing agent for the bowel and for intraperitoneal use as an anti-cancer agent Despite enthusiastic advertisements by th manufacturers, there is no clinical evidenc that this particular agent is of any value and in the experimental animal it has no only been found to be relatively ineffectiv against some animal tumors, but in other has actually increased the incidence of successful implantation.

We have been interested in the problem of contamination of the operative wound b tumor cells, and on the basis of implantatio studies using a number of different anima tumors have found nitrogen mustard to b highly effective, with only moderate interference with wound healing[24]. In general w introduce 50 ml. of HN2 (5 mg. per 100 m of 9 per cent saline) into the bowel lume prior to excising the neoplasm. Followin anastomosis, the wound is irrigated wit the same concentration of the drug and ap proximately 10 mg. is placed intraperitoneally. At times the latter is introduced through an intraperitoneal catheter on the day following operation.

The value of this topical chemotherapeutic agent has been most difficult to appraise. We have seen patients in whom peritoneal contamination has undoubtedly followed transection of the tumor, and in whom following the use of this agent there has been no evidence of recurrent disease. At the same time we have seen other patients in whom, despite the use of the same concentration, there has been local peritoneal implantation. Finally, the use of this agent seems to be associated with a higher incidence of abdominal wound dehiscence as well as breakdown at the site of the bowel anastomosis. For these reasons one must be very cautious in the use of these drugs, and only further studies will determine whether the benefits gained will justify the apparent increase in morbidity as far as wound-healing is concerned.

The presence of neoplastic cells in peripheral blood and the probable increase in the dissemination of tumor cells during operation have led a number of individuals to advocate adjuvant systemic chemotherapy at the time of surgical treatment. Although such treatment apparently offers some benefit in the management of carcinoma of the breast, there is no evidence of any benefit in lesions of the gastrointestinal tract. In fact both the morbidity and mortality of surgical treatment have been increased in association with the use of these drugs.

New Diagnostic and Therapeutic Pointers

Finally I should like to comment upon a few diagnostic and therapeutic "advances."

1. *Acute dilatation of the colon in association with ulcerative colitis* should be considered as an ominous sign of acute and severe disease. Systemic toxicity is usually evident, and characteristically the lesion is manifested by a tender but soft abdomen. Tympany is present and peristalis absent. Ulcerations are frequently deep, and bleeding may be prominent. In this condition perforation is common and the mortality is high. When dilatation from this cause is observed, a decompressive colostomy, cecostomy or resection is advisable.

2. *Temporary gastrostomy.* Just as a temporary gastrostomy has made the patient undergoing upper abdominal surgery more comfortable and reduced morbidity from ileus, similar, though fewer, indications exist in surgery of the large bowel. Patients who are poor risks, who have had massive surgical procedures, or in whom prolonged ileus may be anticipated are excellent candidates for a temporary gastrostomy using a no. 16 Foley cateter. We prefer to suture the visceral peritoneum to parietal peritoneum with interrupted Lembert sutures of no. 4-0 silk at the site of gastrostomy. The catheter can be removed after six or seven days. Only in severely debilitated patients has the gastrostomy wound failed to closed immediately.

3. *Polyps of the colon and rectum.* Because of their possible relationship to cancer, an extremely agressive attitude toward the indentification and removal of colonic polyps has been advocated during the past few years. This approach has included coloscopy during operation, extended colotomy, and even subtotal colectomy as methods of diagnosis and treatment. I believe the trend is somewhat more conservative today. This can be attributed to (1) the question of whether such polyps ever develop into cancer; (2) the recognition of the morbidity and mortality of this aggressive approach; and (3) the significance of a "juvenile" polyp. In one series of 311 patients undergoing colotomy and polypectomy, 149 had endoscopic examinations through one or more incisions. Postoperatively, 14 per cent of the subjects had complications directly related to colotomy—namely, superficial wound infection, paracolic abscess, fecal fistula, bleeding, and ileus. Corrective operations were necessary in 7 per cent of cases. Infected wounds were three times as frequent among patients having colonoscopic examination as among those in whom the instrument was not used. All intraperitoneal abscesses and fecal fistulas occurred in patients in whom polyps were located with the sigmoidoscope[25].

Although juvenile polyps occur primarily in children, they are also seen in adults and have a characteristic histologic pattern. Grossly they resemble the adenomatous polyp, but appear to have a granular surface. They are probably inflammatory in origin and cannot be considered premalignant. As a consequence, such polyps in children, if asymptomatic and beyond the reach of a sigmoidoscope, do not constitute an indication for celiotomy and polypectomy.

The villous adenoma is an interesting variant of colonic polyps in that it can excrete large amounts of *potassium*, leading to severe diarrhea, electrolyte depletion, metabolic acidosis, and azotemia. These adenomas have a characteristic appearance and location. Because of the incidence of local invasion, a more aggressive surgical approach is advised.

Summary

Some of the changes in the surgical management of colonic diseases occurring during the past decade have been reviewed. Due perhaps to the nature of the problem as well as to the incidence of the disease, the most conspicuous alterations have been those in the management of diverticular disease and carcinoma. Although resection of the colon affected by diverticulitis has become a relatively safe procedure, the morbidity remains relatively high. This fact, coupled with the inherent nature and course of the disease, warrants a fairly conservative attitude toward the problem.

Despite the overwhelming emphasis on adjuvant chemotherapy in the surgical treatment of cancer, the results are much more impressive in animals than in human beings. This fact may well be related not only to the inadequacy of chemotherapeutic agents, but to the frequent lack of correlation between the response of tumors in the experimental animal and of those occurring in man. Again, a conservative and some what skeptical attitude must be adopted.

Finally, there are a number of techniques which have improved not only our diagnostic acumen but also some of our surgical methods of treatment.

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Valves of the Posterior Urethra in Infants

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Since valvular obstruction in the posterior urethra was first recognized by Langenbeck[1] in 1802, the condition has been the subject of widespread interest in the field of surgery. Not until 1913, however, was the first case clinically recognized and surgically treated, by Young[2], who reported it to the Johns Hopkins Medical Society in November of that year.

Incidence

Although the true incidence of the condition is not known, many authors consider it to be quite rare. Only 204 reported cases were collected from the literature (by McCrea[4]) in 1949, but subsequent reports indicate that the condition is much more common[5-8]. Torp[5], in 1954, reported 4 cases observed in infancy at autopsy in a six-month period, while Raper[6], in 1953, observed 7 cases in a 20-month period in Leeds, England. Williams[7a], in 1954, encountered 13 cases in a two-year period at the Hospital for Sick Children in London. Kjellberg and Ericsson[8], in 1957, reported 52 patients with valvular obstruction; in 12 the findings did not warrant surgery. Only 8, or 20 per cent, of those operated on were less than 6 months of age.

The present report covers 3 cases seen in a babies' hospital in this country during an 18-month period. A review of the literature since 1950, including these 3 cases, discloses a higher percentage of patients less than 6 months of age. This indicates to me a growing awareness and earlier recognition of the condition, which are our only hope for better management.

Pathogenesis

Valves in the posterior urethra are of congenital origin. The etiology has been thoroughly studied by Tolmatschew[10], Bazy[11], Lowsley[12], Watson[13], Glenister[14], and Williams[7a], but no one theory satisfies all types of valves included in the generally accepted classification of Young and MacKay.[15]. In brief, the valves are formed *in utero,* and have been observed as early as the 50 mm. stage (Williams[7]). If they persist, they constitute an obstruction to the outflow of urine from its initial formation at the third month of gestation. It is known that the fetus voids urine into the amnionic fluid[16]. Valvular obstruction impedes the voiding mechanism, resulting in hypertrophy of the bladder, and hydroureter and hydronephrosis. This mechanism operates for as long as six months before birth, when the clinician first has an opportunity to examine the patient. Unless the condition is recognized early and the proper corrective measures are instituted, it progresses to uremia and total renal decompensation. When the diagnosis is finally stumbled upon, it is often too late. "Stumbled upon" is used intentionally, as many of these patients are treated for gastrointestinal complaints or anemia for weeks or months before the true diagnosis is made.

Diagnosis

Early diagnosis depends upon awareness of the condition, in conjunction with a careful history and physical examination. The history of these little patients depends upon the observations of the parents or nurse. Anyone who has noticed a normal male infant void knows he can do so with considerable force, projecting a stream often of 2 or more feet. Also, a normal male infant doesn't stay wet all the time. Infants with urethral valves do, as there is an overflow type of incontinence, with constant dripping. When the infant does void, it is as a dribble or very weak spouting of only some 2 inches. All infants in this small series and those in the recent literature presented a palpable bladder[5-7].

Frequently the kidneys, and at times the ureters, are also palpable. Urethrocystographic findings are not the primary factor in the early diagnosis of this condition, as Friesbach, Waterhouse and Mellens[17] recently suggested, but are confirmatory. Urethrocystography will demonstrate the valve beautifully[8], and also will show trabeculation of the bladder and vesicoureteral reflux when present. The diagnosis rests on the history and physical examination.

The management of these patients always requires supportive therapy, which frequently includes preliminary cystostomy drainage prior to correction of the obstruction. Even in the newborn infant, the obstruction has been present for six months, and the non-protein nitrogen level is elevated. Corrective surgery must be deferred until the blood chemistry values approach normal and the patient is in the optimal condition for surgery. We were able to prepare only 1 of our 3 patients for operation, as the other 2 succumbed. One died from massive atelectasis shortly after birth, and the other from septicemia resulting from a perinephric abscess at the age of 12 days.

Case Reports

Case 1

A full-term white male infant weighing 8 pounds 6 ounces was observed to be cyanotic at birth. Administration of oxygen was started immediately, and he was given 300,000 units of Dicrysticin, with repetition of the dose in 12 hours. Physical examination revealed complete atelectasis on the right, and eight hours after birth the distended bladder was noted. Before urologic consultation could be obtained, the patient died of respiratory failure at 13 hours of age. Autopsy disclosed that death resulted from congenital atelectasis. The kidneys and ureters were found to be markedly enlarged and the bladder thickened and trabeculated, secondary to valvular obstruction in the posterior urethra.

Case 2

A 5 pound 8 ounce white male, born four weeks prematurely, was referred to the hospital 12 hours after birth because of cyanosis and of crying upon straining to void, with passage of only a drop or two of urine. Physical examination revealed a palpable bladder extending almost to the umbilicus, and bilaterally palpable kidneys.

On admission the non-protein nitrogen was 75 mg. per 100 ml. and the hemoglobin 17.9 Gm. The white blood cell count was 17,800, with 73 polymorphonuclears, 19 lymphocytes, and 3 juvenile forms. The urinalysis after catheterization, which yielded 90 cc. of clear urine, showed a trace of albumin, an occasional white blood cell, and a specific gravity of 1.006. A no. 10 Foley catheter was passed only after no. 8 and no. 10 sounds were passed, and was left indwelling. The child was given 5 mg. of Furadantin four times a day and 300,000 units of Dicrysticin every 12 hours. He improved for four days, during which his urinary output ranged from 270 to 400 cc. every 24 hours, with a comparable intake of fluids. On the seventh day, however, the temperature began climbing and continued to spike to 103 F. for the next four days in spite of supportive therapy and oxygen. The child died 10 days after admission.

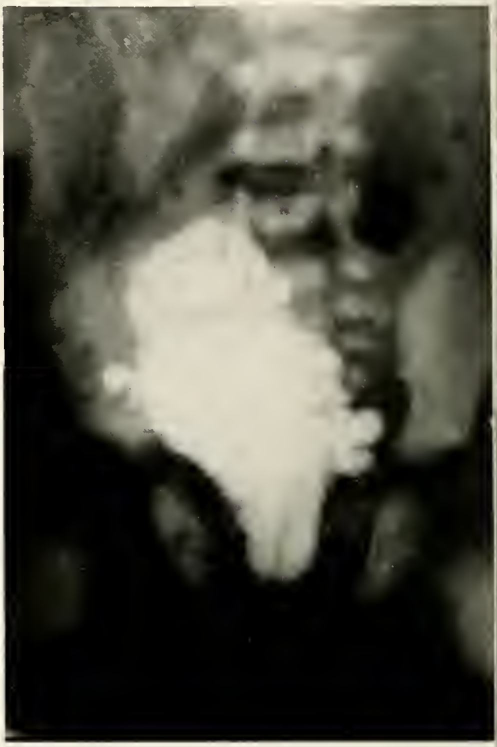

Fig. 1. Roentgenogram showing a greatly dilated prostatic urethra. The valve appears as a negative shadow. The bladder is markedly trabeculated.

An autopsy revealed a perinephric abscess on the left, which was reported as the cause of death, with right bronchopneumonia as a contributing factor. Examination of the urinary tract revealed, in addition, bilateral hydronephrosis and hydroureters, with a thickened bladder and a distended prostatic urethra secondary to valves distal to the trigone (fig. 1). If this could have been salvaged for operation, there was adequate renal tissue remaining to sustain life and permit recovery.

Case 3

A 7 pound 8 ounce white male was admitted to the hospital at 1 month of age because of fever and vomiting of 48 hours' duration. He had eaten

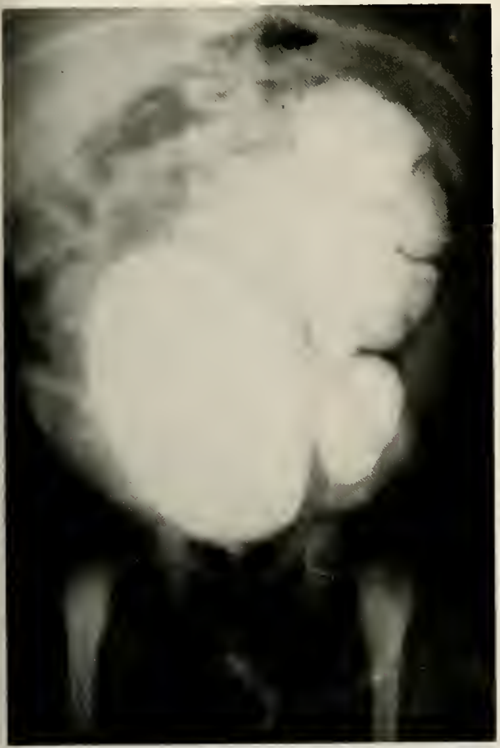

Fig. 2. There is massive reflux to the left kidney, which fills the entire left side of the abdomen. The ureter has a greater diameter than the small bowel.

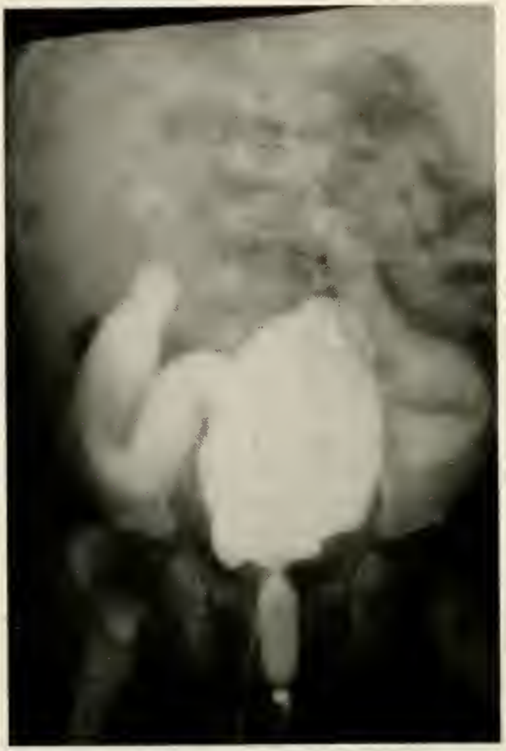

Fig. 3. Roentgenogram showing marked dilation of the prostatic urethra associated with contracture of the bladder neck. There is also bilateral vesicoureteral reflux.

poorly since birth and stayed constantly wet. He had seemed to have colic almost constantly at night, with frequent spells of crying. The non-protein nitrogen was 74 mg. per 100 ml. and the hemoglobin 9.9 Gm. The white blood cell count was 7,500, with 49 per cent polymorphonuclears, 39 lymphocytes, and 12 band forms. The urine contained an occasional white blood cell, showed a trace of albumin and a specific gravity of 1.003.

Physical examination revealed an acutely ill child with a bladder distended to the umbilicus. A catheter could not be passed. A cystostomy was done, with the removal of 70 cc. of clear urine, and the bladder was decompressed over the next 24 hours. The total urinary output was 520 cc. in 24 hours. The patient was given three transfusions of 50 cc. of blood during the next week, and 48 hours after operation began taking his formula. He was given 19 mg. of Furadantin four times a day during the next three weeks, during which the non-protein nitrogen fell to 42 mg. per 100 ml. and the daily output of urine ranged from 330 to 500 cc. He was discharged 20 days after operation, weighing 7 pounds 14 ounces and having a hemoglobin of 13.5 Gm. A suprapubic tube was in place and draining well. The child had not been observed to void while in the hospital. A cystogram revealed massive ureterovesical reflux (fig. 2), and a trabeculated bladder with a dilated posterior urethra (fig. 3).

Six weeks after the cystostomy the child weighed 8 pounds 10 ounces. The hemoglobin was 12.5 Gm. and the non-protein nitrogen 34.5 mg. per 100 ml. A transpubic excision of the prostatic valves was performed, as advocated by Bross[18], and at the same time a wedge of the bladder neck was excised. A urethral catheter was inserted, but the patient pulled it out on the second day. The cystostomy tube was left in place. The patient's course was uneventful except for fever spiking to 102 F. for 48 hours following operation. His discharge from the hospital was delayed five days because of the development of thrush, which responded to Mycostantin. He was discharged two weeks following operation, with the wound healed and the suprapubic tube draining well. The non-protein nitrogen was 32 mg. per 100 ml. Two weeks later a cystogram revealed a more normal appearing prostatic urethra (fig. 4). Two and a half months after operation there was still reflux, but less trabeculation.

The child has continued to gain (present weight 18 pounds), and eight months postoperatively shows continued improvement of his upper urinary tract (fig. 5). Reflux persists, however, and the suprapubic tube is still in place. The specific gravity is 1.014, the non-protein nitrogen is 28 mg. per 100 ml., and the urine contains no albumin. It is contemplated that the tube will be kept in place for possibly six more months. An intravenous pyelogram does not give satisfactory concentration, and the urethra accommodates a no. 10 sound easily.

Discussion

The problem of urethral valves is a discouraging one. In the 3 cases reported here, 2 of the patients died before operation, and the third is still being treated. The only hope lies in earlier diagnosis by the pediatricians who first see these patients. Management includes intensive supportive treatment, usually necessitating cystostomy drainage, followed by operation when possible. The transpubic approach is preferred in infants befause it provides better exposure and direct excision of the obstructing tissues. Also, it is not always practical, even with a urethrotomy, to use an infant resectoscope in a male infant in the early months of life. Frequently there is associated bladder neck hypertrophy which can and should be corrected at the same operation. In the majority of the cases reported the literature, the patients treated by transurethral surgery were past the age of 3 months. Gross[15] prefers the transpubic approach even in older patients, because it holds less danger of incontinence, and provides more adequate exposure and more thorough excision of the offending tissues.

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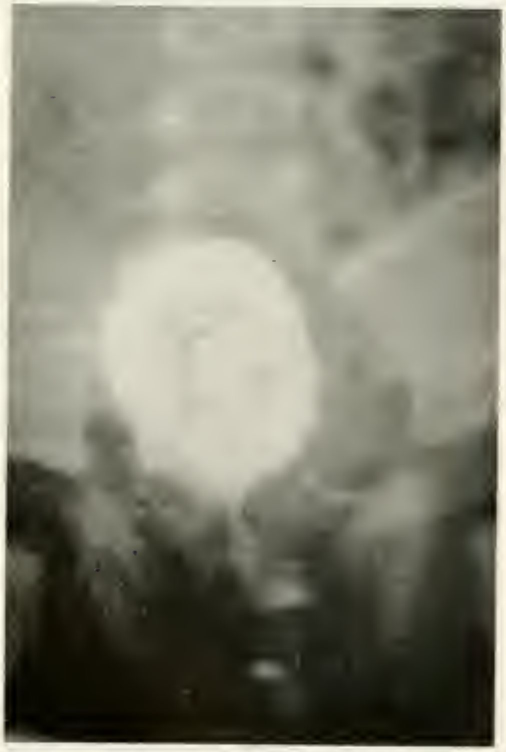

Fig. 4. Four weeks after operation the prostatic ureter appears more normal and the bladder neck is nicely funneled.

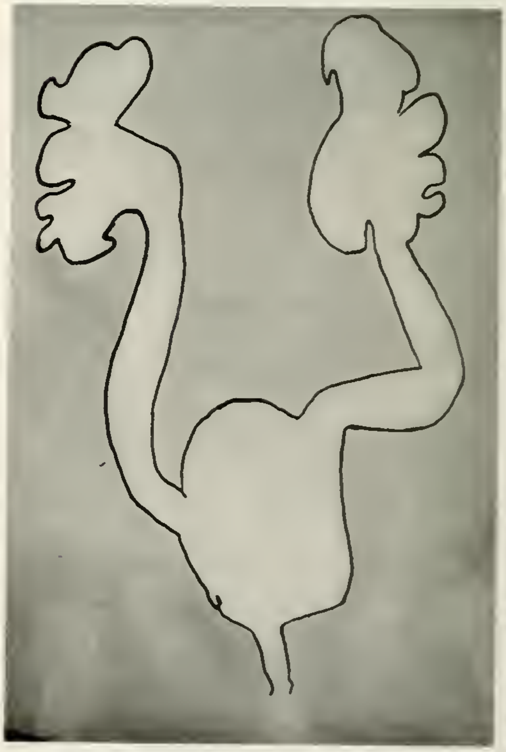

Fig. 5. Eight months after operation the upper tracts have improved, but are far from normal. The cystostomy tube is still in place.

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Long-Term Management of Atherosclerotic Heart Disease

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Coronary atherosclerosis is our leading public health problem, ranking first in incidence and mortality. The atherosclerotic process begins in childhood and progresses to narrowing of the coronary artery in the young adult, as vividly noted in U. S. soldiers killed in the Korean War[1]. Because of the duration of the disease process, the long-term control of patients with clinical coronary atherosclerosis, angina, coronary insufficiency, and/or myocardial infarction is of as much importance as the relief of the acute symptoms.

In the hospital cases reviewed, 15 to 30 per cent of patients with myocardial infarction died in the first two months. However, the mortality in private practice patients having their first infarction without complications is 3 to 5 per cent[2]. In general, patients with myocardial infarction without complications during or after the infarction have a death rate of 5 per cent per year, but this figure is doubled in the presence of persistent chest pain, congestive failure, severe arrhythmia, thromboembolic phenomena, cardiomegaly, or shock[2]. The deaths are largely due to cardiac causes[3]. In most series[3, 4], 70 per cent or more of the patients return to the same or other productive employment after their first infarction.

These statistics merely point out the magnitude of the number of patients involved in a consideration of long-term therapy.

Weight reduction in the obese patient with symptomatic atherosclerosis goes without saying. Evaluation of all patients for diabetes, hypertension, and hypocholesterolemia, in which the presence of atherosclerosis is more marked, is essential. Control of these conditions is obviously of prime importance.

This discussion will deal with the long-term medical management as opposed to treatment of the acute symptoms of coronary atherosclerosis. The categories to be considered are rehabilitation, diet, drugs for control of hyperlipemia, and long-term anticoagulant therapy. Since time does not allow a discussion of all the methods used, only those principles that apply to the majority of cases will be considered.

Rehabilitation

Long-term management should begin as soon as the acute symptoms have been controlled. Discussing with the patient and his family the good prognosis and likelihood of returning to work eliminates much of the cardiac neurosis. After the initial period of

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three to four weeks, prescribed exercises should be started. Walking has been advocated as the best form of physical activity[5], and can be increased every five to six days until a total of 1 to 2 miles can be tolerated in a day.

Convincing proof of the ability of the post-infarction patient to carry out almost any type of work is available[6], and should be discussed with him during his convalescence. A thorough knowledge of his job requirements allows the physician to evaluate carefully the amount of physical and emotional stress encountered each day. The use of the Master two-stet test[7] and/or the Vansalva maneuver[8] will help to determine his capabilities if there is any question of his physical endurance. These studies are best performed two and one half to three months after the attack. Emotional stress has been shown to increase blood coagulation, the serum cholesterol level, and myocardial consumption of oxygen[9]. The emotional problems can usually be controlled or improved by discussing them with patient. The importance of total rehabilitation cannot be overemphasized[10].

Dietary Measures

The study of diet in relation to the mortality of coronary atherosclerosis reveals an increased death rate in groups living on food high in animal protein, fats, and total calories[11]. Groen's[12] study of serum lipids in Trappist monks, who are vegetarians, and Benedictine monks, who eat a varied diet, shows the effect of dietary intake in groups essentially the same in other respects.

Study of various medications and diets used to retard or possibly reverse the atherosclerotic process has been difficult. Because of the variation of symptoms and signs in a given individual, many years are required to evaluate any form of therapy. The atherosclerotic study group at the Bowman Gray School of Medicine has evaluated many types of food substances and drugs in pigeons which developed atherosclerotic lesions spontaneously. It is well known that all pigeons are hyperlipemic, but only certain species develop atherosclerosis. Of these, the atherosclerotic lesions that occur in one species, without alteration of their normal diet, are macroscopically and microscopically like those in humans[13].

Virtually total elimination of fats from the diet is apparently not as beneficial as the substitution of poly-unsaturated fats for saturated fats[14]. Clarification of the terms "animal" and "vegetable" fats, or "saturated" and "unsaturated" fats seems advisable. Not all animal fats consist of saturated fatty acids, nor do all vegetable fats contain poly-unsaturated fatty acids, which have the ability to decrease serum cholesterol. In essence, the consumption of fats that cause a reduction in the serum cholesterol and decrease the atherosclerotic process are ideal. Poly-unsaturated fatty acids such as linoleic, linolenic and arachadonic, and the highly unsaturated fatty acids of fish oil seem to accomplish this best[14 15]. The evaluation of the cholesterol and the atherosclerotic index of the aorta in the pigeons studied clearly illustrates the value of fats high in these polyunsaturated fatty acids[13]. The unsaturated vegetable oil, safflower oil, proved superior to other fats in reducing serum cholesterol and the degree of atherosclerosis of the aorta.

Increased knowledge among the lay public of the relationship of cholesterol and atherosclerosis has stimulated the drug and food manufacturers to produce many items designed to "lower cholesterol." Many of these products are of questionable value, though they have been sold in vast amounts. Giving a patient small amounts of unsaturated fatty acids in capsules three or four times a day seems absurd. To obtain an adequate amount of unsaturated fat by these methods would require 60 to 90 capsules a day, at a cost of $65.00 to $135.00 per month[16]. The use of unsaturated vegetable oils, such as safflower and corn oil, is much cheaper and seems to be more effective.

Drugs for the Control of Hyperlipemia

Many other ways of lowering the serum cholesterol have been explored. The difference in incidence between men and women has led to the use of estrogen. Continuous administration of this substance does lower the blood lipid content[17], but the secondary feminization has made it unpopular in men. The value of estrogen in the 40 to 50 year old man and the menopausal woman manifesting the complications of atherosclerosis has shown promise[14]. The use of thyroid has waxed and waned, but has never had wide acceptance in the United States. It seems ironic to increase the basic metabolic rate in a patient with coronary atherosclerosis, but we must still await its long-term effect on the atherosclerotic process. Nicotinic acid, in doses of 4 to 6 Gm. per day has been shown to reduce serum cholesterol[18]. This finding has also been noted by those studying this drug at Bowman Gray. The side effects of flushing usually disappear with continued use, but the more recently reported impairment of hepatic function in approximately 45 per cent of the cases at the Mayo Clinic[19] necessitates very close observation of the patient and discontinuance of the drug at times.

MER-29 has been shown to block the synthesis of cholesterol, with a reduction in the serum level[20]. There is a corresponding increase in desmosterol, but seemingly a lowering of the total blood lipids. The question of whether the decrease of cholesterol by merely blocking it at the final step of synthesis actually decreases the atheromatous process has not been answered. Lowering the serum cholesterol level alone seems to be of questionable value in the prevention of atherosclerotic lesions. In spite of a significant decrease in serum cholesterol with the use of Vanadyl sulfate, the progression of the atheromatous process was equal to that noted in control pigeons[13].

Of the factors influencing coronary atherosclerosis, nutrition is the most amenable to correction, according to Stamler[21]. Diet is probably the best, and certainly the least expensive, method of controlling the blood lipid level. Maintaining the patient on polyunsaturated fats in amounts sufficient to lower the total fat to 20 per cent of the total caloric intake seems ideal. This diet should be followed by anyone having significantly elevated lipid levels or evidence of ischemic heart disease. Utilizing the fat tolerance test, it has been noted that the patient with ischemic heart disease has markedly more prolonged hyperlipemia after the ingestion of fatty meals[22]. Dietary control of fats in these patients certainly seems warranted. Control of other dietary foods is still being discussed widely, but no definite conclusions have been reached.

Anticoagulants

With the general acceptance of anticoagulant agents in the treatment of acute myocardial infarction, their use in the long-term therapy of coronary atherosclerosis has been advocated[23]. In 1947 Nichol and Fassett[23a] established the feasibility of this therapy and, with others[23b], demonstrated its effectiveness. Thomas and others[23d] have shown the superiority of continuing rather than discontinuing anticoagulant therapy after its use in the treatment of acute infarctions. A significant decrease in the recurrence of thromboembolic phenomena and myocardial infarction was noted in those having continuous anticoagulant treatment, although their death rate was only slightly less. Friedburg[23h] found a reduction in mortality of one-third to one-fourth, and a decrease in the recurrence of myocardial infarctions of one-fifth to one-sixth, in patients receiving anticoagulant agents as compared with the controls. These patients were followed for approximately two years. British investigators[24] found that continuous anticoagulant treatment reduced the recurrence rate to one-fifth that of controls for the first two years after infarction in men under 55 years of age.

In general, the use of long-term anticoagulant therapy after myocardial infarction decreases the rate of subsequent infarctions, thromboembolic phenomena, and, in most series, the annual mortality. The benefit is more marked in men under 60 years of age and in those who have more than one infarction.

Evidence is accumulating to support the use of anticoagulants in patients with progressive angina and coronary insufficiency[25]. Nichol[25a] believes that patients with impending myocardial infarctions should be treated promptly and continued on long-term anticoagulants. Many of his patients obtained relief of angina, and only 6.6 per cent had an infarction.

The problems involved in long-term anticoagulation have been discussed[26]. The availability of proper laboratory facilities and the reliability of the patient are the two most important factors. Hemorrhagic diathesis, ulceration, or neoplastic lesions of the gastrointestinal tract; hepatic dysfunction; reduction in urinary excretion, and personality disorders are usually contraindications[26a]. Generally, the patient on a fixed dose of an anticoagulant in the hospital can be checked every week for one or two months. The interval between prothrobin determinations can then be gradually lengthened to three or four weeks. He should be warned about the use of large amounts of salicylates, broad spectrum antibiotics, and even phenobarbital, as these drugs may precipitate bleeding episodes. Vitamin K should be kept by the patient, but used only after consulting his physician. Cards stating that the patient is taking an anticoagulant are available for him to carry in case of injury. They provide identification and facilitate the immediate administration of vitamin K when necessary.

Summary

1. The vital importance of the rehabilitation of patients with coronary atherosclerosis is discussed, in view of the fact that 70 to 75 per cent should return to the same or another useful job.

2. The superiority of diets utilizing polyunsaturated fatty acids as a means of controlling blood lipids is discussed. The use of capsules containing unsaturated fatty acids is thought to be too expensive and of only questionable value. Other means of lowering the serum cholesterol are mentioned.

3. Under the proper circumstances the use of long-term anticoagulant agents seems advisable in patients with increasing angina, coronary insufficiency, and/or myocardial infarction.

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Huntington's Chorea

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Among those illnesses of man that are capable of producing unlimited disability and ultimate death but at the same time are preventable, Huntington's chorea stands out as a prime example. This bizarre ailment is not as rare as might be expected by practicing physicians who see only one or two cases in a lifetime. Its potential for becoming more prevalent justifies the present report.

The psychiatric aspects as well as other long drawn out disabling features of Huntington's chorea cause many of its victims eventually to be consigned to mental institutions. At present the medical and social service staffs of Dorothea Dix Hospital in Raleigh are compiling data on the cases which have come to all units of the state hospital system, in order to get a clearer picture of the true prevalence of the disease in North Carolina. Follow-up data on the relatives and children of these patients are also planned. For example, since 1933, 21 cases of Huntington's chorea, confirmed on the basis of family histories, clinical findings and autopsies, have been recorded at Dorothea Dix Hospital.

Social histories obtained from the families of the 21 patients indicate that there were 96 living relatives when the histories were taken, some of whom were beginning to manifest symptoms of the disease. In view of the reproductive capacities of these relatives, this figure must be considerably above the total of 117 cases and relatives on whom we have records at the present time.

Genetic Characteristics

Huntington's chorea is inherited by way of a Mendelian dominant gene. Since transmission is not dependent on sex, 50 per cent of the children of a gene-carrying individual

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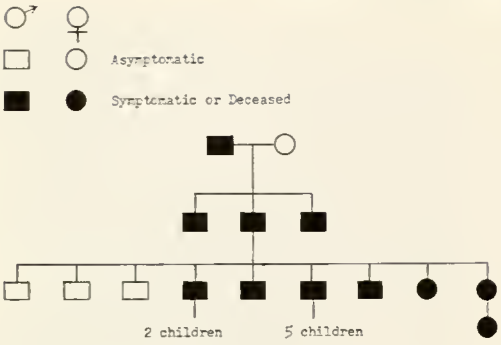

Figure 1

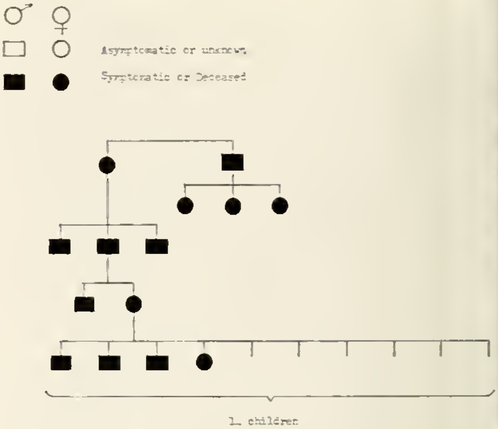

Figure 2

will become affected. As others have pointed out[1], these cases often appear in localities where members of affected families remain, marry, and have children. We know of two such areas in eastern North Carolina, and at least one in the western part of the state. In one of these areas the following family history was obtained.

Three brothers and one sister died of Huntington's chorea at Dorothea Dix Hospital. The father, paternal grandfather, and two uncles also had had the disease. Of five siblings, two are known to be affected. One of the brothers had five children, another two, and the sister, though unmarried, had a daughter who died of the disease. This family is shown schematically in figure 1.

From another locality we have the record of three brothers and a sister who had the disease. In this family, however, the Mendelian gene had been transmitted through the mother, whose grandmother—that is, our patient's great-grandmother—had died of Huntington's chorea, as had three of the grandmother's nieces. Our patient's grandfather carried the gene, as did two of his brothers and one of his sons. Thus the great-grandmother transmitted the disease to a son, who passed it on to a granddaughter, and later to several great-grandchildren who in turn died with it at the State Hospital. There were six other siblings in this family group on whom we have as yet no follow-up data, and 14 children who are now being investigated. These 14 are the great-great-grandchildren of the ancestor mentioned above. A relative of the family is now in the hospital with Huntington's chorea, and this patient has nine siblings who are now being investigated. This family is shown schematically in figure 2. The disease almost resembles a parasite in its ability to propagate itself.

It was once thought that all cases of Huntington's chorea in this country could be traced through Boston to Lincolnshire, England. In 1630 Puritans migrated to Scotland and also to Boston. Actually, however, all ethnic groups and races are affected[1].

An individual who has inherited the trait will inevitably succumb to the disease or to one of its complications unless some other illness or accident intervenes. Unfortunately he has no way of knowing whether or not he carries the trait, as the symptoms do not become manifest until about the age of 37. By the time the diagnosis is made, he may have married and had children. The average duration of life after onset is about 13 years[2].

It is an interesting fact that if all affected individuals were to have no children, Huntington's chorea would disappear in one generation. The value of disseminating this information to the families in question is obvious. With the extensive research being done in the field of genetics these days, it will be interesting to watch for possible developments in Huntington's chorea, particularly in reference to predicting the onset of symptoms from a study of the chromasomal structure of affected individuals.

Signs and Symptoms

The signs and symptoms of the disease are well known. It is worth emphasizing that the first indications may appear in the form of personality disturbances. For example, 6 patients were admitted to Dorothea Dix Hospital with behavioral problems characterized by violence, depression, confusion, vagrancy, prostitution, paranoid thinking, and suicidal ideas and attempts.

Fifteen patients were admitted with definite neurologic signs which led to the diagnosis shortly after admission. These symptoms had not been apparent to the referring physician in 6 cases, nor to the hospital personnel on admission in 2 cases. Most of the patients, however, gave histories ranging from 2 to 12 years of behavioral changes characterized by depression, overactivity, hallucinations, violence, confusion, and lack of interest in personal appearance and surroundings, and were admitted to the hospital when their families became unable to cope with them. One case was diagnosed on admission as alcoholism without psychosis, and another as catatonic schizophrenia. The true picture of Huntington's chorea developed later. Thus a personality disturbance in a person having a family history of Huntington's chorea should alert physicians to the course of events to follow.

The uncontrollable movements of these patients are quite characteristic and usually begin with the facial muscles—grimacing, nodding the head, moving the jaw and later the extremities, thus making the diagnosis more obvious. These movements diminish when the patients are quiet, and disappear during sleep, but become very marked during excitement. Many of our patients grimace and grit their teeth almost continuously. The muscular movements are gross, not fibrillatory, and often assume a jerking quality. It is interest to observe how often a jovial nature is apparent in spite of the extremely disabling movements.

Treatment

Treatment of this condition is largely symptomatic. Unfortunately little help is available for the mental aberrations. A variety of drugs and neurosurgical procedures have afforded varying degrees of help in controlling the hyperkinesis and abnormal movements. Reserpine, given intravenously and orally for as long as 7 days, has been reported by Lazarte and others[3] to help reduce the hyperkinesis and impairment of voluntary motor functions. Procaine amide, reserpine, BAL, and Meratran seem to be helpful in some cases (Forrest[4]). Pallidal coagulation and thalamotomy, along with related procedures, have opened up new therapeutic and experimental approaches to this and other diseases associated with abnormal movements (Spiegel and others[5], and Guiot[6]). We have found that Dartal sometimes helps reduce the violent jerking of some patients, and chlorpromazine quiets some of the more agitated ones.

Terminally, patients with Huntington's chorea present extremely difficult management problems. The movements become totally uncontrollable, and self-inflicted abrasions and decubiti may develop. Nutrition and fluid balance may be disturbed, and pneumonia — often on the basis of aspiration—may be a terminal event.

Autopsy may reveal little morphologic change in the brain. There may be varying degrees of atrophy, especially in the frontal and sylvian regions, ventricular dilatation, and basal glandular disease[7].

A recent case report by Facon and others[8] showed that at autopsy the lesions are not limited to the striatum, but are distributed in many other areas of the brain, such as the cerebellum, substantia nigra, locus caeruleus, dorsal paramedian nucleus, and subthalamic regions adjacent to the third nucleus and to the third ventricle, as well as demyelination in the medial portion of the mediolemiscus. It would appear that the lesions may be quite diffuse.

Conclusion

Huntington's chorea is more prevalent in North Carolina than is generally realized. With the current emphasis on preventive medicine, we would like to add this disabling and eventually fatal disorder to the list of preventable diseases and plead for a greater dissemination of information about it to patients and their families and to the medical profession in general.

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A Report on Renovist - A New Intravenous Pyelographic Medium

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The value of intravenous pyelography in modern urologic diagnosis is established. Employing present-day techniques, a successfully completed intravenous pyelographic examination usually provide definitive information regarding the state of the renal tract and gives some indication of the patient's renal function. A major factor contributing to the success of this procedure is the availability of a number of contrast agents which may be safely administered and which provide a sharp, clear outline of the entire renal system. Notable among those agents which have been extensively employed are Miokon*, Hypaque† and Renografin‡[1-5].

Recently, a new intravenous medium, Renovist (Squibbs), was introduced for clinical appraisal. This agent is a modification of the Renografin formulation designed to provide contrast of equivalent density but with lower viscosity to facilitate administration. It is an aqueous solution of the methylglucamine and sodium salts of 3,5- diacetylamino-2,4,6-triiodobenzoic acid (diatrizoate) containing approximately 37 per cent iodine or 3.72 grams of iodine per cubic centimeter. Over the past few months Renovist has been routinely administered in intravenous pyelographic studies at this hospital. It has proved to be a satisfactory medium, with minimal unwanted reactions, as evidenced by the following brief report.

Material and Method

Four hundred and fifteen consecutive and unselected patients were examined with Renovist. On admission all these patients presented symptoms suggestive of urinary tract disorder. Many complained of acute abdominal pain. Some had been injured in automobile accidents and others presented gunshot wounds. Because of the acute nature of the conditions seen, many patients were examined without ideal preparation for urography. Seven of the 415 patients in the series had had previous pyelographic examinations with other contrast agents. A total of 46 of the 415 patients under study gave a history of seasonal hay fever or of other mild allergic manifestations.

Except in emergency cases where preparation was impossible, each patient was routinely prepared for examination by purgation and dehydration during the preceding 18 hours. Immediately before examination, 1 cc. of Renovist was administered intravenously to each patient to determine sensitivity; if no reaction was observed within 1 minute, the full dose was given over a period varying from 2 to 4½ minutes, and serial films were exposed at 5, 10, 15, and 20 minutes after injection. Two different doses of Renovist were given—20 cc.

From Mercy Hospital, Charlotte, North Carolina.

*Brand name for diprotrizoate sodium (Mallinckrodt Chemical Works).

†Brand name for diatrizoate sodium (Winthrop Laboratories).

‡Brand name for diatrizoate methylglucamine (E. R. Squibb & Sons).

was administered to 119 of the 415 patients and 25 cc. to 296 patients.

The resulting pyelograms were judged and classified on the basis of the quality of contrast and the extent of opacification of the urinary tract displayed in the films. "Very good" films provided satisfactory visualization of the entire urinary tract, including calyces, pelves, and ureters, as well as the urinary bladder, all in one or more single films. "Good" films provided incomplete visualization of the ureters on a single film, but satisfactory delineation of the entire renal system was obtained by a composite examination of serial films in the particular case. Films classified as "fair" were those in which calyceal visualization was not ideal, or in which ureters were not completely visualized. "Unsatisfactory" films provided such poor outline of the urinary tract that they were not of diagnostic quality.

Results

The quality of films obtained for the whole series of patients examined with Renovist was as follows:

Table 1
Results

Amount Injected	**No. Patients**	**Quality of Films**			
		Very good	**Good**	**Fair**	**Unsatisfactory**
20 cc.	119	43	57	15	4
25 cc.	296	194	73	24	5
Totals	415	237	130	39	9

"Good" or "excellent" films were obtained in 367 of the 415 patients, while the films were rated as only "fair" in 39 cases and "poor" in 9 cases. Of the 367 patients who obtained satisfactory films, 125 were examined on an emergency basis without ideal preparation. On the other hand, 13 of the 39 patients with films of only "fair" quality were among those who had been poorly prepared, and only 2 patients who had received suitable preparation before examination obtained unsatisfactory films.

There were three principal causes for the poor quality of films obtained in some patients in this series or for failure of the examination in others. These were:

1. Failure to administer the full dose of Renovist because of small or inadequate veins or because of reaction to the medium.
2. Obstruction of the urinary tract due to stones, prostatic hypertrophy, or primary renal diseases of various sorts.
3. Poor preparation of the patient.

Of 7 patients examined previously with other media, the films with Renovist were considered to be of equal quality in 2 cases, and superior in the other 5. One patient who had experienced a reaction to the previously administered medium had no reaction with Renovist. Four patients in the series obtained satisfactory films even though they received only 5, 12, 15, and 18 cc. of Renovist, respectively.

Side Effects

There were no serious reactions to Renovist in any of the 415 patients examined, and 398 had no reaction whatsoever. Some unwanted effects were observed in 17 patients, but in only 3 of the 17 were they of any clinical concern (the reactions in these patients were nausea and urticaria). The intensity and frequency of reactions was less when the period of administration of the medium exceeded 3 minutes. It is of interest that the only patients who developed hives were among those receiving the medium at a slow rate of injection. It certainly cannot be said, however, that any relationship between the rate of injection and the occurrence of a sensitivity reaction of this type was apparent. Arm pain and bitter state, reported by several patients when injected more slowly, did not occur when Renovist was administered in 2.5 minutes or less.

The reactions experienced by 17 patients in this series during examination with Renovist are shown in table 2.

Comment

Renovist proved to be a very satisfactory medium for intravenous pyelography in this series of patients. The films obtained were diagnostic in the great majority of cases, even in some patients who were considered to be poorly prepared. This finding is of special interest at this hospital, which

Table 2

Reactions

Reaction	No. of Patients	Times of Onset
Nausea	6	5 to 20 min. after injection
Nausea and vomiting	1	On completion of injection
Arm pain	2	During injection
Bitter taste	2	During injection
Burning sensation in arm and chest, with bitter taste	1	During injection
Coughing	1	After 15 cc. had been given
Sneezing	1	After injection
Urticaria	2	10 and 40 min. after injection
Sensation of warmth	1	During injection

is situated in an area with a high incidence of urinary tract stones, many patients being necessarily examined during acute attacks without time for prior preparation. Furthermore, a number of patients are admitted literally "off the streets," often with emergency conditions requiring immediate examination.

An injection of 20 cc. of Renvoist was completely adequate in most cases, since "good" or "excellent" films were seen in 100 of the 119 patients (84 per cent) examined with this quantity. The diagnostic quality of films obtained in 4 cases following administration of only part of the full dose indicates that in cases where a full dose of the medium cannot be given, the examination should not be terminated without taking at least one or two films.

Reactions were mild and relatively few, and even some of those reported, such as bitter taste and arm pain, may have been due more to suggestion than to drug.

Summary

Renovist, a new radiopaque medium, has been administered in doses of 20 or 25 cc. to 415 consecutive and unselected patients as the contrast agent for intravenous pyelographic examination. The acute conditions presented on admission necessitated examination without ideal preparation in 125 cases. The urograms obtained following administration of Renovist permitted a definite diagnosis in 367 of the 415 patients (88 per cent), while films in an additional 39 cases provided helpful diagnostic information. The films were of poor quality in only 9 of the 415 examinations performed. While more than half of the films considered to be only "fair" were found among those who had been poorly prepared, many examinations of patients without prior preparation were rated as "very good" or "good."

There were no serious reactions to Renovist in any patient. Some reactions were observed in 17 of the 415 examined with the medium, but in only 3 of the 17 were they of clinical concern, these being nausea and urticaria. The reactions that occurred during examinations with Renovist included nausea or nausea and vomiting, arm pain, bitter taste, sensation of warmth, coughing, sneezing, and urticaria.

Renovist proved to be a very satisfactory contrast agent for use in intravenous urography, and was associated with only minimal reactions.

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Report from The Duke University Poison Control Center

JAY M. ARENA, *Director*

BARBITURATE

Barbiturates are a leading cause of acute poisoning, either through error (often in a patient already befuddled by sedatives), suicidal intent, or unguarded exploration by children. The patient is found stuporous or comatose, often with severe respiratory depression and cyanosis. A rough measure of the depth of poisoning is shown by the response to talking, shouting, pinprick, and squeezing of the Achilles tendon. If the degree of coma is fairly light—that is, if the patient responds readily to noxious stimuli —recovery is virtually assured if proper treatment is instituted.

The most threatening state is one of total areflexia, unresponsiveness to other stimuli, unremitting coma, and signs of impending circulatory failure as well as apnea. A patellar reflex can, however, be elicited in some apneic patients, and the corneal reflex may be absent in some who are lightly narcotized. The vital signs—color, respiratory rate, pulse, blood pressure, and body temperature —are the critical guides to management and outlook.

A first requirement is to insure a clear airway, if the patient manifests cyanosis, labored breathing with retraction of the neck or intercostal muscles, wheezes, rhonchi, or large amounts of secretion in the pharynx. A simple immediate measure is to extend the patient's head backward while pulling the angle of the jaw forward. If this is successful, insertion of an oral airway will probably be adequate for breathing purposes. If these maneuvers do not relieve respiratory obstruction, suction must be employed, passing a nasal catheter as far as it will go into the trachea. Absence of a cough reflex at the level of the larynx upon passage of the catheter means that intubation must be performed at once, preferably with a cuffed endotracheal tube and with the expert assistance of an anesthesiologist. Use of the endotracheal tube must then be continued until vigorous coughing returns; if more than 48 hours elapse, a tracheotomy is necessary.

Once an airway has been insured, and if no more than a few hours have passed since ingestion of the barbiturate, gastric lavage is indicated. If endotracheal intubation has been done, there is no danger of aspiration. Otherwise, lavage is performed with the patient carefully positioned on one side, his face downward over the edge of the bed.

Deeply comatose patients should receive 100 per cent oxygen, 4-6 liters per minute by nasal catheter. Respiration should be assisted if necessary, and the apneic patient should be placed in a respirator. When artificial respiration is used, there is some danger that acidosis or alkalosis will develop as a result of hypo-or hyperventilation, and it would be ideal to measure the blood carbon dioxide and pH at intervals if the means are at hand.

The deeply comatose patient will undoubtedly require fluid and nutrition administered by vein for one to three days. An infusion should be started promptly, and pressor agents such as levarternol, phenylephine, or norepinephrine added if the blood pressure is low. Small amounts of potassium salts may also be added after the first day of intravenous feeding if there is adequate urinary output. Volume expanders or whole blood may be required if shock, anemia, or hemorrhage (as from the digestive tract) become evident. As a rule, antibiotics should be given as a safeguard against pneumonia. Unquestionably the patient should be kept warm; the fear of exaggerating the oxygen requirements is probably less valid than the effort to restore normal physiology (particularly when oxygen is being supplied).

Finally, assiduous nursing care is needed. The patient is best kept with the head low so the secretions can drain from the respiratory tract, suction is employed as necessary, and the patient is moved about in bed at regular intervals. If coma is prolonged, an indwelling catheter may be practicable. If renal function is severely depressed because of hypotension and the central antidiuretic action of barbiturates, ex-

Outline of Management

Pathophysiology	**Treatment**
Overdosage of barbiturate, if taken orally	Immediate gastric lavage, using warm water
Respiratory center depression with slow, shallow breathing; cyanosis often present	A. Respiratory stimulants such as picrotoxin or pentamethlyenetetrazol, administered in subconvulsive doses B. Artificial respiration C. Administration of 100 per cent oxygen
Circulatory depression and shock due to depression of vasomotor center, as well as direct action on smooth muscle in blood vessel wall	A. Pressor amines such as levarterenol (which act directly on vascular smooth muscle) B. Blood transfusions C. Trendelenburg position
Water loss from skin and lungs; decrease in urine; electrolytes variable	A. Adequate hydration with 5 to 10% glucose in water to facilitate renal elimination of barbiturates B. Use of electrolytes based on analysis of plasma
Hypostatic pneumonia resulting from hypotension and hypoventilation	A. Prophylactic antibiotics

change transfusions, peritoneal dialysis, or the artificial kidney may be life saving. Removal of barbiturates from circulating blood can also be achieved in this fashion. Digitalis should be given for heart failure, and aspirin suppositories can be given by rectum for the hyperthermia.

Not included among the foregoing measures are the variety of so-called analeptic drugs and barbiturate antagonists which have been propounded, each in its turn as more or less specific antidotes for barbiturate poisoning. Their chief value would be to stimulate the medullary centers and so restore respiration and vasomotor tone. There is no general agreement that this is achieved, and there is also a hazard of causing convulsions with their use, thus increasing oxygen requirements in a hypoxic patient.

Current practice in most centers which have had considerable experience with barbiturate poisoning is to regard immediate establishment of adequate pulmonary ventilation as well as control of shock as of prime importance, while analeptics are considered subsidiary if not actually contraindicated. Some clinicians still favor, for deeply narcotized patients, the supplemental use of amphetamine, caffeine, and picrotoxin or Metrazol. The evidence indicates that the most favorable results—that is, lessened mortality and morbidity—are obtained by paying careful attention to respiratory and circulatory function.

The above outline of the management of acute barbiturate poisoning may be helpful.

> We should realize that the practice of medicine is governed principally by "statistics," and we should be delighted when we have good statistical data to guide our treatment of the individual patient. Our problem is not that there is too much statistics in medicine, but that there is not enough. In too many areas reliable statistics are not available. As a result, we don't really know how to treat individual patients. In such cases, we must follow our hunches and do the best we can, recording the outcome carefully and objectively so that we may eventually add our accumulated experience ("statistics") to the body of medical knowledge.—Editorial: Statistics Are All Very Well, But . . .: New York State J. Med. 60: 3599 (Nov. 15) 1960.

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IS BIG SCIENCE RUINING SCIENCE?

More than a year ago John Russell created much controversy by an article in *Harper's Magazine* suggesting that too much money was being spent on scientific research, and that it was wrong to assume that answers to any research problem could be found if only enough money were spent. Mr. Russell's position is given powerful backing by the Director of the Oak Ridge National Laboratory, Dr. Alvin M. Weinberg, in a talk given before the American Rocket Society and published in *Science* for July 21.

Dr. Weinberg begins with an analogy between the Pyramids, the Sphinx, and the great temple at Karnak and our big science achievements. Just as they were built to express the aspiration of the Egyptian rulers, our huge rockets and high-energy accelerators were built to add to our country's prestige.

Dr. Weinberg raises three questions: (1) "Is Big Science ruining science?" (2) Is Big Science ruining us financially?" (3) Should we divert a larger part of our effort toward scientific issues which bear more directly on human well-being than do such Big Science spectaculars as manned travel and high-energy physics?"

Dr. Weinberg thinks there is real danger that Big Science will ruin true science by nurturing publicity in order to obtain public support; by spending money instead of thought; and by the great increase in the number of administrators. He thinks there is grave danger to our universities from the invasion of Big Science. He points out that a professor of science should be extremely well qualified as a scientist and teacher, but that if he becomes too involved with Big Science, he will have to become a public relations man and administrator.

Next, Dr. Weinberg discusses the question "Is Big Science ruining us financially?" by pointing out that our research and development budget has increased 10 per cent a year—much more rapidly than has the gross national product. If this rate of increase were maintained, he estimates that in about 65 years we would be spending all our money on science and technology. He recommends that the rate of increase of this budget should be drastically reduced.

In discussing the final question, Dr. Weinberg admits that we must use scientific achievement as a means of competing with the U.S.S.R., but that we do not have to make manned flight into space the primary event in these scientific Olympic Games. He argues against concentrating on space flight on three grounds—hazard, expense, and relevance. He states that there is far more hazard from radiation in a space flight than was even suspected until recently. A man would need to be shielded from the radiation hazard if he landed on the moon. Dr. Weinberg aptly says that "The Lord provided His children with a marvelous radiation shield, the atmosphere, and He did not intend them to poke their heads into His unshielded reactors." The expense of sending a man even to the moon would be so gigantic as to stagger the imagination.

Finally, he would rather choose issues which have much more bearing on the world as a part of man's every-day environment and part of man's welfare. Doubtless almost everyone will agree with him that "most Americans would prefer to belong to the society which first gave the world a cure for cancer than to the society which put the first astronaut on Mars."

His conclusion is that "Those cultures which have devoted too much of their talent to monuments which had nothing to do with the real issues of human well-being have usually fallen upon bad days . . . we must not allow ourselves, by short-sighted seeking after fragile monuments of Big Science, to be diverted from our real purpose, which is the enriching and broadening of human life."

* * *

FIFTIETH ANNIVERSARY OF THE NORTH CAROLINA PUBLIC HEALTH ASSOCIATION

Sincere, even if belated, congratulations are extended to the North Carolina Public Health Association and the Guilford County Health Department for having attained their golden anniversary, and for having accomplished so much within a half century.

The Guilford County Health Department was the first county unit in the state, and one of the first in the nation. Quite fittingly, the June issue of the *Health Bulletin* in honor of the occasion, is a golden anniversary issue.

Miss Grace Daniel of Salisbury, president of the North Carolina Public Health Association, presided over the General Session of the Annual Meeting. Dr. E. H. Ellinwood, Health Director of Guilford County, was the host. Dr. E. G. McGavran, Miss Lula Belle H. Rich, and Miss Daniel contributed to make this a memorable issue. Governor Terry Sanford issued an official statement on June 16, designating the week of June 25 to July 2 as Public Health Week in North Carolina.

FLORENCE NIGHTINGALE'S INFLUENCE LIVES ON

Many years ago a British surgeon, Stephen Paget, wrote a little classic, *Confessio Medici*, largely for young doctors. In it he said that he could not wish for a young doctor just beginning practice anything better than an operation, a serious illness, or both —for the experience would make him a better doctor. That a similar wish might apply to hospital administrators was illustrated in his book, "Hospital, Doctors and Dollars," written by the editor of *The Modern Hospital*. A fracture sustained from a skiing accident gave him the opportunity to study hospital routine *in vivo* after years of observing hospital phenomena *in vitro*. His two chief complaints were about being waked at daybreak to have his temperature taken, and the "barbarous schedule" used in serving meals.

He did not offer a theory for the century-old custom of waking hospital patients at daybreak when they have nothing to do all day but to stay in bed—so here goes for a plausible explanation that has long intrigued this editor: That barbarous custom began with Florence Nightingale, the mother of modern nursing. She began her hospital experience during the Crimean War. It was customary to have an army waked early in order to begin the day's operations. Miss Nightingale trained her nurses to wake patients early: in turn they trained theirs, and each succeeding generation of nurses has followed, without stopping to think, Florence Nightingale's original teaching.

Another custom for which Florence may or may not have been responsible is that of serving the last meal of the day in the middle of the afternoon. The stock excuse given for this barbarism is that it is necessary in order to let the hospital help get away as early as they want to. One cannot help wondering how successful hotels and restaurants manage to keep their help overtime. Certainly, it would seem that hospital administrators should consider the comfort of the patient more than the convenience of the employee.

President's Message

H.R. 4222

The House Ways and Means Committee wound up its hearings on H.R. 4222 after nine days of testimony. The American Medical Association was ably represented by Dr. Leonard W. Larson, Dr. Edward R. Annis, Dr. Ernest B. Howard, and other physicians from a number of state medical societies.

Our own Dr. Hubert Poteat, representing the North Carolina State Medical Society, appeared at the committee hearing at 10:00 A.M. on August 1, through the efforts of Mr. Harold Cooley, the representative from Dr. Poteat's congressional district. All these gentlemen made very fine presentations, and were well received by the committee. Dr. Poteat did a perfect job before this committee, as he does before the state committee on medical legislation. We are very grateful to him for the time and effort spent for us.

Other than physicians who appeared before the House Ways and Means Committee in opposition to H.R. 4222 was H. Lewis Reitz, representing the life and health insurance industry. He told the committee that insurance company actuaries estimate that the benefits provided by H.R. 4222 would cost $2.2 billion in 1963—more than twice the estimate made by the Department of Health, Education and Welfare.

Other points made by opponents of the bill are as follows:

"Dr. Annis pointed out that although the opening sections of the bill disclaim control over the practice of medicine or the administration of hospitals, specific provision in H.R. 4222 'provides the means by which federal officials *can* regiment and control all providers of the services covered.' . . . He also declared that the proposal would be 'staggeringly expensive,' and that the Department of HEW cost estimates are 'based on limited experience, chronic optomism and an unwillingness to admit that the program would be severely overutilized.' . . . Other points made by Dr. Annis were that H.R. 4222 would: (a) endanger the entire Social Security System; (b) undermine private health insurance; (c) discourage voluntary health agencies; (d) cover millions who neither need nor want help; (e) destroy the concepts of individual and family responsibility; and (f) eliminate the states' historical responsibility in the health care field."

The general consensus, according to Dr. Poteat and others who appeared before the committee, is that the bill will not be enacted this year, but that the medical profession will have to stay on the alert for the future.

We have the best system of health care in the world, and there is not a single person in North Carolina in need of medical care today who cannot obtain it. I have never known a person in my own county who was turned down for hospitalization or medical care regardless of his financial condition.

CLAUDE B. SQUIRES, M.D., F.A.C.P.

Bulletin Board

COMING MEETINGS

Duke University School of Medicine, Symposium on the Commonwealth of Children—Duke University, October 4-8.

University of North Carolina School of Medicine, Postgraduate Courses in Medicine—Asheville, beginning October 3 and continuing for six weeks; Morganton, beginning October 4 and continuing six weeks.

North Carolina Fifth District Medical Society Meeting—Carolina Hotel, Pinehurst, October 11-12.

Association of American Physicians and Surgeons, Annual Meeting—Grove Park Inn, Asheville, October 12-14.

The Charlotte Postgraduate Seminar—Charlotte, October 18-19.

North Carolina State Board of Medical Examiners—Sedgefield Inn, Greensboro, October 27.

North Carolina Neuropsychiatric Association, Annual Meeting—Raleigh, October 27.

University of North Carolina School of Medicine Symposium—Chapel Hill, November 9-10.

North Carolina Pediatric Society, Annual Meeting—Sedgefield Inn, Greensboro, November 10-11.

North Carolina Academy of General Practice, Annual Meeting—Carolina Hotel, Pinehurst, November 26-29.

First National Congress on Medical Quackery—Washington, D. C., October 6-7.

Southeastern Allergy Association, Annual Meeting—Charlottesville, Virginia, October 21.

Southern Medical Association, Annual Meeting—Adolphus Hotel, Dallas, Texas, November 9-10.

American Medical Association, Clinical Meeting—Denver, Colorado, November 27-30.

Southern Surgical Association, Annual Meeting—The Homestead Hotel, Hot Springs, Virginia, December 5-7.

New Members of the State Society

The following physicians joined the Medical Society of the State of North Carolina during the month of July:

Dr. Robert Seitz Cline, Graham Street, Box 707, Warrenton; Dr. Ruth Peschel, Duke University Hospital, Durham; Dr. Roston Meldrim Williamson, 306 South Gregson Street, Durham; Dr. Shankar Nath Kapoor, 306 South Gregson Street, Durham; Dr. Adrian Verwoerdt, 2216 Elba Street, Durham; Dr. William Lord London, 306 South Gregson Street, Durham; Dr. Thomas DeArman Kinney, Duke University Medical Center, Durham; Dr. Robert Alvin Keppel, 1319 7th Street, N. W., Hickory; Dr. Donald Clifford Hartzog, Jr., 2069 Elizabeth Avenue, Winston-Salem; Dr. John Stover Gaskin, Jr. 265 North 3rd Street, Albemarle; Dr. Kenneth Daand Hall, Duke University Medical Center, Durham; Dr. James Edward Stephens, Medical Center Building, Asheville; Dr. Robert Perry Crouch, Medical Center Building, Asheville; Dr. Edward Kenneth Ishey, Jr. 409 Flatiron Building, Asheville; Dr. Thomas Herbert Collawn, Presbyterian Hospital, Charlotte; Dr. Raymond Thomas Doyle, 130 South Kings Drive, Charlotte; Dr. Thomas Leroy Dulin, 200 Greenwich Road, Charlotte; Dr. Fitz Gerald Hiestand, Jr,. 1350 Kings Drive, Charlotte; Dr. Donald Swift Kellan, Jr., 1500 Elizabeth Avenue, Charlotte; Dr. William Davie Lyday, 1350 Kings Drive, Charlotte; Dr. Thomas Neely Massey, Jr., 217 Travis Avenue, Charlotte; Dr. Charles Frederick Heinig, 1822 Brunswick Avenue; Charlotte, Dr. Sara Lynn Bailey, 1705 Fountain View; Charlotte; Dr. James William Skaggs, Jr., Box 36366, Derita Branch, Charlotte; and Dr. H. E. Mattox, Jr., Carolina General Hospital, Wilson.

News Notes from the University of North Carolina School of Medicine

Three North Carolina Medical School graduates have taken top honors during their internships at the George F. Geisinger Hospital in Danville, Pennsylvania.

Drs. John Young of Lexington, Dr. William B. Abernathy of Chapel Hill, and Dr. William Huff of Charlotte, were cited for the "best overall work during the intern year." Each received a cash prize from the hospital.

* * *

The University of North Carolina School of Medicine was recently awarded a research program-project grant by the National Heart Institute, U. S. Public Health Service, for the establishment of a comprehensive center for the study of hemorrhage and thrombosis, Dr. Walter Reece Berryhill announced recently.

The grant was awarded for a period of seven and one-half years in the total amount of $1,568,-435. It will allow the expansion of the well known research programs on bleeders, thrombosis, and blood coaqulation which have been carried on in the School's Departments of Pathology and Physiology for many years. This expansion will allow for comprehensive coverage all the way from basic biochemical, physiologic, and pathologic studies through clinical investigative studies on affected patients.

The grant will be used for two main purposes (1) to support a three-bed clinical research unit in the North Carolina Memorial Hospital, and (2) to strengthen the laboratory work in the Department of Pathology.

* * *

Sixteen faculty members of the University of North Carolina School of Medicine recently received promotions to positions ranging from full professor to assistant professor.

Promoted to full professor were Dr. Charles

E. Flowers, professor of obstetrics and gynecology; Dr. Charles D. Van Cleave, professor of anatomy; Dr. Colin G. Thomas, professor of surgery; Dr. Carl W. Gottschalk, professor of medicine and physiology and recipient of the American Heart Association Career Investigatorship.

Appointed to the rank of associate professor were Dr. Walter Hollander Jr., associate professor of medicine and director of the Clinical Research Unit; Dr. Thomas F. Williams, associate professor of medicine and preventive medicine; Dr. John H. Schwab, associate professor of bacteriology; Dr. Robert H. Wagner, associate professor of pathology in pathological chemistry and biochemistry.

Promoted to assistant professorships were Dr. John C. Herion, assistant professor of medicine; Dr. William G. Wysor, assistant professor of medicine; Dr. Thomas C. Gibson, assistant professor of medicine; Dr. Dan A. Martin, assistant professor of medicine and preventive medicine; Dr. Cornelius Lansing, assistant professor of psychiatry; Dr. Aubrey G. Tolley, assistant professor of psychiatry; Dr. Robert L. Timmons, assistant professor of surgery; Dr. John L. Simmons, assistant professor of surgery.

* * *

Claude McClure, Jr., M.D., and Everett Hoyle Schultz, M.D., have been appointed to the faculty of the University of North Carolina School of Medicine.

Dr. McClure, a native of Dungannon, Virginia, was appointed as assistant professor. He received the Ph.D. degree in 1960 at the University here. He received the B.S., and M.D. degrees at Wake Forest College. He served as an instructor at Duke University from 1953-1956, and was an associate at the University of Tennessee during 1956-1957.

Dr. Schultz, a native of Winston-Salem, was appointed an associate professor. He received the M.D. degree at Wake Forest College, Bowman Gray School of Medicine. From 1955-57, he was an assistant in radiology in the North Carolina Memorial Hospital. Among other positions held by Dr. Schultz are assistant in radiology at Massachusetts Institute of Technology and assistant professor at the University of Florida Teaching Hospital.

* * *

Dr. Edgar T. Thompson, president of the North Carolina Society for Crippled Children and Adults, has announced that the Easter Seal Research Foundation has awarded a grant of $8,925 to Dr. T. Franklin Williams, associate professor of medicine at the University of North Carolina, as principal investigator, and Dr. Mary B. Arnold, assistant professor of pediatrics, as associate investigator.

The grant is to be used for a research project in studies of phosphorus and calcium metabolism, with particular reference to renal excretion of phosphorus under various conditions, particularly vitamin D resistant rickets.

* * *

Some 25,000 physicians saw a prize-winning University of North Carolina School of Medicine exhibit at the national convention of the American Medical Association in New York City. The exhibit, on telecardiography, was designed by William Thornton of Faison, rising third year student of the U.N.C. School of Medicine. A first-prize winner at a recent Student A.M.A. convention in Chicago, it was shown at the national A.M.A. convention by invitation.

* * *

Scientific Director Dr. William A. Jeffers of The Life Insurance Medical Research Fund said recently that the University of North Carolina School of Medicine has received a grant of $20,900. The money is to be used by Dr. Carl E. Anderson to study the chemistry and metabolism of plasmalogens.

A national television audience recently followed a day in the life of Dr. A. John Bambara, a staff physician at North Carolina Memorial Hospital here.

Dr. Bambara has been on the hospital house staff for 18 months, engaged in postgraduate studies in psychiatry. The program was filmed at the site of his former practice in Flemington, New Jersey, over a nine-month period.

Sponsors for the show, which was televised by the National Broadcasting Company, were the American Medical Association and Merck, Sharp and Dohme.

* * *

Two postgraduate courses in medicine, sponsored by the University of North Carolina School of Medicine, will begin in October in Asheville and Morganton.

They will consist of two lectures one day a week over a six-week period. The Asheville course, which begins October 3, is co-sponsored by the Buncombe County Medical Society; and the Morganton course, which begins October 4, is co-sponsored by the Burke County Medical Society.

All Asheville lectures will be given in the Buncombe County Medical Society Library at Memorial Mission Hospital at 5 and 7:15 p.m.

The afternoon Morganton lectures will be given at the Nurses' Home of Grace Hospital at 4:30 p.m. The 7:30 p.m. lectures will be given at the Mimosa Golf Club.

The lecturers for these courses, in order of their appearance are: Dr. Herman Hellerstein, Western Reserve School of Medicine; Dr. Luther Talbert and Dr. Loren MacKinney, both of the University of North Carolina School of Medicine; Dr. John Siegling, Medical College of South Carolina; Dr. Charles Vernon, University of North Carolina School of Medicine; Dr. Charles Howell, Jr., the Bowman Gray School of Medicine; Dr. F. William Sunderman, Jefferson Medical College.

Both courses are acceptable for credit by the American Academy of General Practice for the number of hours attended by the individual physician.

* * *

Miss Margaret L. Moore, assistant professor and director of curriculum in physical therapy of the University of North Carolina School of Medicine, has been elected National Secretary of the American Physical Therapy Association. The election took place at the Thirty-eight annual convention of the Association, held in Chicago.

News Notes from the Bowman Gray School of Medicine of Wake Forest College

September 10, 1961, marked the twentieth anniversary of the Bowman Gray School of Medicine.

A banquet commemorating the occasion was held Saturday, September 9, 1961. Dr. Ward Darley, executive director of the Association of American Medical Colleges, spoke.

Dr. Harold W. Tribble, president of Wake Forest College, presided at the banquet, which was attended by approximately 250 prominent educators, laymen, and friends of the institution.

Courses primarily designed to prepare students for the study of medicine were instituted at Wake Forest College in 1887. The college was, therefore, among the first institutions in the United States to recognize the need for proper premedical training.

A School of Medicine was established in 1902. It was renamed the School of Medical Sciences in 1937 and operated as a two-year medical school until 1941, when it was moved from Wake County to Winston-Salem as a four-year medical school in association with the North Carolina Baptist Hospital.

In the past 20 years, the medical school has made significant gains in the size of the faculty, in the facilities for teaching and research, and in the amount of research and training funds.

The medical school had a faculty complement of 74 in 1941. The faculty now numbers 210. There are now 210 members of the student body.

Physical facilities have multiplied almost threefold—from 70,465 square feet to more than 191,000 square feet. Research and training funds have risen from $62,525 to more than $1½ million.

* * *

Dr. Alanson Hinman, assistant professor of pediatrics and director of Amos Cottage at Graylyn, has announced that 10 beds are available at the cottage for the admission of eligible children.

Amos Cottage is a service division of the Bowman Gray School of Medicine for the study and training of educable mentally retarded children.

North Carolina State Board of Medical Examiners

The North Carolina State Board of Medical Examiners will meet at the Sedgefield Inn, Greensboro, on Friday, October 27, at which time applicants for license by endorsement of credentials will be interviewed.

American Hearing Society

An emphatic warning to anyone faced with a loss of hearing to consult an otolaryngologist before considering the purchase of a hearing aid is contained in a new Public Affairs pamphlet, "You and Your Hearing," published recently.

Written by Dr. Norton Canfield, a distinguished ear specialist, with the cooperation of the American Hearing Society and the Royal Neighbors of America, the pamphlet points out that there are many types of hearing defects and that many of them can be overcome by treatment of surgery without the use of a hearing aid. The pamphlet is available for 25 cents from the Public Affairs Committee, Inc., 22 East 38th Street, New York 16, New York.

American Medical Association

First National Congress on Medical Quackery

The First National Congress on Medical Quackery, which will be held in Washington, D. C., October 6-7, was conceived by the American Medical Association's Department of Investigation.

Recognizing that medical quacks and charlatans thrive in the dark shadows of public ignorance, the Department felt the meeting would be an effective way of throwing the spotlight on this problem.

The National Congress will be co-sponsored by the Food and Drug Administration, which quickly accepted an invitation by the American Medical Association to join in the venture.

State and county medical societies were urged by the A.M.A. House of Delegates in June to send representatives to the National Congress, which will be a springboard for a vigorous, multi-faceted attack on medical quackery.

American Association of Medical Assistants

New wonder drugs, medical quackery, future training programs, and professional liability are among subjects to be considered by medical assistants when they gather October 13-15 at Reno, Nevada, for the fifth annual convention of the American Association of Medical Assistants.

Additional information may be obtained from the A.A.M.A. headquarters, 510 North Dearborn Street, Chicago 10, Illinois.

OCCUPATIONAL HEALTH CONGRESS

The American Medical Association will hold its twenty-first national Congress on Occupational Health in Denver, October 2-4.

To be held at the Brown Palace Hotel, the three-day meeting is sponsored by A.M.A.'s Council on Occupational Health in cooperation with the Colorado State Medical Society.

The congress serves as a meeting for the formal presentation of papers on occupational health as well as a forum in which occupational health problems and questions can receive the attention of acknowledged experts in this field.

A high light of the meeting will be the presentation at the annual banquet of the award to a physician selected by the President's Committee on Employment of the Physically Handicapped for outstanding contributions to the welfare and employment of the nation's physically handicapped.

AMERICAN THORACIC SOCIETY

The American Thoracic Society invites submission of abstracts of papers relating to the general field of tuberculosis and other respiratory diseases for presentation at its Fifty-Seventh Annual Meeting to be held in conjunction with that of the National Tuberculosis Association in Miami Beach, Florida, May 20-23, 1962.

Abstracts must be in the hands of the program committee not later than January 5, 1962. Eight copies should be submitted. Each abstract should be limited to 300 words.

Further information regarding the submission of abstracts may be obtained by writing Asher Marks, M.D., chairman of the Medical Sessions Committee, American Thoracic Society, 1790 Broadway, New York 19, New York.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination (Part I), written, will be held in various cities of the United States, on Friday, January 5, 1962.

Current Bulletins may be obtained by writting to Robert L. Faulkner, M.D., Executive Secretary and Treasurer, 2105 Adelbert Road, Cleveland 6, Ohio.

Diplomates of this Board are urged to notify the office of the executive secretary and treasurer of a change in address.

CALEB FISKE FUND

The trustees of America's oldest medical essay contest, the Caleb Fiske Prize of the Rhode Island Medical Society, announce two subjects for this year's dissertation, open to any doctor of medicine in the nation, for which a cash prize of $500 will be awarded. The subjects chosen are: "Recent Advances in the Treatment of Malignant Disease," and "Current Status of Cardiac Surgery." An essay on either subject must be typewritten, double spaced, and should not exceed ten thousand words.

Essays must be submitted by December 11 to the Secretary, Fiske Fund, Rhode Island Medical Society, 106 Francis Street, Providence 3, Rhode Island.

NATIONAL SOCIETY FOR MEDICAL RESEARCH

Dr. Hiram E. Essex was elected president of the National Society for Medical Research at a meeting of the society held on June 29, in New York. Dr. Essex is emeritus professor of physiology and former chairman of the Section on Physiology of the Mayo Foundation for Medical Education and Research. He succeeds Dr. Lester R. Dragstedt, of the University of Florida.

Other officers elected are, vice presidents, Dr. Ward F. Darley, executive director of the Association of American Medical Colleges, and Dr. John B. Youmans, Director of the Division of Scientific Activities of the American Medical Association; and secretary-treasurer, Dr. W. T. S. Thorp, dean of the College of Veterinary Medicine of the University of Minnesota.

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

In view of the rapid expansion which is forecast in the use of nuclear energy, x-rays, and other sources of radiation, a greatly expanded program for research grants in the field of radiologic health has been developed. These grants are offered to support research by individuals, universities, hospitals, laboratories, and other public or private institutions in the assessment and control of manmade and natural radiation exposures to the individual, no matter how the separate components may originate.

For information and/or application forms please address Dr. Paul F. Hahn, Chief, Office of Extramural Grants, Division of Radiological Health, U. S. Public Health Service, Washington 25, D. C.

VETERANS ADMINISTRATION

In view of the evidence on virus origin of cancer in animals, it now would be difficult to assume that cancer in humans is of different origin, an authority in this field of research said recently.

In a new book written for scientists and physicians, Dr. Ludwik Gross of the Bronx, New York, Veterans Administration hospital and Sloan-Kettering Institute said it now is quite apparent that most of the malignant tumors of chickens and the great majority of the malignnant tumors of mice are caused by viruses.

In recognition of his outstanding research on leukemia and malignant tumors, Dr. Gross has just been awarded the Walker Prize of the Royal College of Surgeons of England, at Oxford University.

NATIONAL ASSOCIATION OF BLUE SHIELD PLANS

Dr. Leonard W. Larson, President of the American Medical Association, will be one of the featured speakers at the Blue Shield Annual Program Conference to be held October 23-24, 1961 at the Drake Hotel in Chicago, under sponsorship of the National Association of Blue Shield Plans.

BLUE CROSS ASSOCIATION

Walter James McNerney, director of the Bureau of Hospital Administration, School of Business Administration, University of Michigan, has been named president of the national Blue Cross Association.

Professor McNerney succeeds James E. Stuart, who became executive vice president of the Association in 1959 and president in 1960, and is now chairman of the board of governors.

Book Reviews

Myxedema. By Jerry K. Aikawa, M.D., 106 pages. Price, $5.00. Springfield, Ill.: Charles C Thomas, Publisher, 1961.

In a recent review of two books Mr. Sidney Harris commented that they both had a severe case of literary edema—"That disease so pandemic in the book publishing business." Dr. Aikawa's book is refreshingly free from this disease. He has put into 99 pages—including references—a fascinating history of myxedema, beginning with T. W. King's recognition of the function of the thyroid gland in 1936. Sir William Gull, in 1875, reported five cases of myxedema, and is credited with being the first to recognize it. Dr. William Ord, in 1878, described the pathology of the condition and first proposed the name **myxedema.** Brief sketches of Drs. Gull and Ord add interest to the story.

Chapter 2 describes the evolution of specific therapy, beginning with transplantation of the thyroid gland. Credit is given Dr. George Murray for first using, in 1891, a thyroid extract in a myxedematous patient, who continued to take the extract until she died in 1919 at the age of 74. Soon after Dr. Murray reported using the extract by hypodermic injection, three physicians reported independently that thyroid extract was just as effective by mouth as by hypodermic injection.

Chapter 3 discusses the use of the basal metabolic rate in the diagnosis of thyroid disease. The following chapters tell of radioactive iodine, proteinbound iodine, and butinal extractable iodine as diagnostic methods which have virtually supplanted the B.M.R. as an index of thyroid activity. For some reason, Dr. Aikawa did not mention the false high readings obtained in individuals with excess iodine in their system, as from a recent cholecystogram, pyelogram, or myelogram.

The remaining chapters are devoted to the connective tissue in myxedema, the relation between the pituitary and the thyroid, auto-immunity, transport mechanism, autothyroid substances, and congenital enzymatic defects.

The concluding chapter ends with the admission, "It must be apparent to the student of medicine that much remains to be learned about myxedema." This reviewer, however, knows of no student of medicine who has summarized so admirably what is known about myxedema.

One sentence in the final chapter is a fitting end for this review: "Notwithstanding the availability of such diagnostic tools, the single most important prerequisite to the diagnosis of myxedema is the awareness by the physician of the existence of this disease, and the high index of suspicion for it."

Textbook of Medicine. Edited by Sir John Conybeare and W. N. Mann. 1008 pages, 62 figures. Price, $10.00. Baltimore: Williams and Wilkins Company, 1961.

The fact that this book has lived through 13 editions since it first appeared in 1929 is evidence that it is good. It has many features to commend it: First is a literary style that characterizes British writing at its best; second, as the authors say in the preface, they have paid special attention to describing treatment in sufficient detail to be of practical value; third, a delightful sense of humor makes the book quite palatable mental food. For example, in regard to the care of the senile patient there is the statement, "The judicious mixture of television and chlorpromazine can be of great value." Finally, the authors do not waste words, and the book is as brief as is consistent with clarity.

A few minor criticisms are offered lest the reviewer be judged as too enthusiastic. Psittacosis is said to be "due to a virus," whereas the **British Medical Journal,** in 1958, transferred the causative agent to the rickettsial group of organisms. Since Tetracyline is recommended as a specific for psittacosis, and since no other known virus is influenced by the antibiotics, this distinction is rather important.

After the first section, headed "Disease Due to Infections," tuberculosis, venereal diseases, and tropical diseases are given equally impressive headings—although these also are generally considered infections.

Pancreatitis is classified as acute hemorrhagic, subacute, and chronic. The subacute form apparently conforms to our conception of edematous pancreatitis, and the statement, "When chronic pancreatitis is suspected, laparotomy must be undertaken," is rather dogmatic.

The tables of heights and weights of males would be confusing to the average American. For instance, the weight for a 25 year old man, 5 feet 9 inches tall, is given as 10 stones 7 pounds. Since a "stone" to an Englishman means 14 pounds, this weight would be translated as 147 pounds.

These minor criticisms are not intended to discount the real worth of the book. It can be highly recommended, both to practicing physicians and to students. At its very reasonable price, it is a real bargain.

Hospitals, Doctors, and Dollars. By Robert M. Cunningham, Jr. 275 pages. Price, $6.50 New York: F. W. Dodge Corporation, 1961.

As the author states in the foreword, this book is critical of hospitals, nurses, and doctors. He devotes far more space to telling what is wrong than what is right, because he believes that the "critic serves society better than the psalm-singer." The work is composed largely of a collection of editorials and articles written by the author over many years. It is well written, quite readable, and stimulating. It gives doctors, nurses, and hospital administrators the opportunity to see themselves as others see them.

The author discusses quite frankly such controversial subjects as the corporate practice of medicine, hospital charges, abuse of Blue Cross and Blue Shield Insurance, political activity of organized medicine, the unionization of nurses and hospital employees, and many other subjects. The titles of the seven chapters give an idea of the book's contents: Washing Professional Linen in Public; Political Means and Professional Ends; Hospital, Doctors, and Dollars; Practice in Plastic Towers; Hospital Labors and Laborers; Some of My Best Friends Are Doctors; and Moments in a Sober Business.

One of the most interesting comments in the book concerns the author's own experience as a patient, after a fracture sustained in skiing. There he learns first-hand what it means to be waked at daybreak and have a temperature taken which is almost certain to be normal; to have meals served "at grotesque hours;" and to learn how much more consideration is shown to the convenience of the hospital help than to the comfort of the patient.

One inconsistency in the book is found in the chapter, "Some of My Best Friends Are Doctors." Frank Lloyd Wright is included along with Morris Fishbein, Charles F. Wilinsky, and Arthur Bachmeyer. It requires more than the average imagination can stretch to think of Frank Lloyd Wright as a doctor.

The book can be recommended as a rather caustic but readable comment on present-day medical care.

For Young Adults Only: The Doctor Discusses Your Personal Problems. By Frank Howard Richardson, M.D. 133 pages. Price, $2.95. Atlanta: Tupper and Love, Inc., 1961.

Dr. Frank Howard Richardson's latest book continues to dispense the same sensible down-to-earth advice that characterizes his earlier ones. His own training as a physician, as a father, and as a citizen has prepared him well to understand and sympathize with young people.

In this reviewer's opinion, Dr. Richardson's latest book is the best of the series. In it he challenges those in their late teens to accept the responsibilities that go with maturity by telling them they are now young adults rather than older children. His ability to see their viewpoint in facing the problem of growing up enables him to give good wholesome advice without preaching. As Dr. L. Nelson Bell says in the foreword: This book answers for children, as well as their parents, in their late teens questions that are vital and . . . without talking down to the younger group or up to their parents."

In the 21 brief chapters Dr. Richardson discusses frankly but sympathetically the chief problems of adolescence: dealing with one's parents, making friends, dating, petting, going steady, drinking, smoking, going to college, and even marriage.

Dr. Bell's comment on the book is a fit ending for this review: "'For Young Adults Only' bears the stamp of a man who knows, loves, and is concerned for young people. Any parent can profit by the author's approach and in reading this book can be led to a new understanding of the one he longs to help but often feels powerless to approach without being misunderstood."

Heredity in Ophthalmology. By Jules Francois. 731 pages with 629 figures including 6 color plates. Price, $23.00. St. Louis: The C. V. Mosby Company, 1961.

Dr. Jules Francois is professor of ophthalmology at the University of Ghent, Belgium. He is well known for publications dealing with numerous hereditary eye diseases, and in particular for his work on the corneal dystrophies. This volume is a translation of the successful French edition entitled **"L'Heredite en Ophtalomolgie,"** published in 1958.

The first 141 pages of text are devoted to an exposition of general principles of genetics, with particular reference to eye diseases. This discussion is brief and to the point, and should serve the needs of the clinician quite well. The section concerning diseases of the eye alone occupies 400 pages, divided into 18 chapters on a topographic basis. A section on ocular manifestations of general diseases occupies 160 pages.

The discussion of specific disease entities usually follows the pattern of a brief definition or description of the condition extensively documented by families and pedigrees reported in the literature, descriptions of associated diseases, and a summary of genetic conclusions that seem justifiable to the author. These discussions seem to be of uniformly good quality from both the clinical and genetic viewpoints. In certain areas of controversy concerning interpretation, Francois usually presents references to arguments on both sides of the question briefly, and states his own opinion in rather definite terms.

A minor weakness is the very limited discussion of interactions of environmental and genetic factors, and lack of emphasis on the usefulness of genetic information in anticipating the development of genetic disease in specific individuals in order to place them under early treatment. The chapter on preventive management deals with only the probabilities of recurrence of hereditary disease and the indications for surgical sterilization, but does not discuss anticipatory treatment and limitation of disability. As the original text was written prior to 1958, it is to be expected that information concerning abnormal chromosome mechanisms in relation to disease syndromes is not included.

There is extensive bibliographic documentation, and each of the major chapters is followed by several pages of references. The reference lists seem to be quite complete through 1956, and occasionally 1957 references are noted. It is unfortunate that the references are listed only by author's name and place of publication, and do not give the titles of the individual articles. From the viewpoint of American readers, the vast majority of the references are to the European literature, and the coverage of English language literature is not so extensive. The subject and author indices are adequate and make the contents easily available for reference.

The appearance of a new monograph inevitably invites comparison with the older books in the same area. The most important older monograph in English is "Genetics in Ophthalmology" by Professor Arnold Sorsby, published in 1951. The new monograph by Francois is more than twice as large, the discussions are more complete and cover more topics, and are much more extensively documented, as well as being more recent by seven years. It seems likely that Francois will replace Sorsby as the standard reference work in this field.

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The Month in Washington

The American Medical Association cited more than 50 reasons why the vast majority of the nation's physicians believe the Administration's medical care program would be "bad medicine for the people of this country."

The A. M. A.'s objections to the proposal were spelled out in a detailed, 91-page printed statement presented to the House Ways and Means Committee by Dr. Leonard W. Larson, president of the A. M. A.

The committee held two weeks of hearings (July 24-August 5) on the Administration proposal (H.R. 4222) which would provide limited hospitalization, nursing home care, and outpatient diagnostic services for social security recipients. The program would be financed by an increase in payroll taxes on workers, employers, and the self-employed.

Dr. Larson declared that the Administration program would force upon Americans a system of health care in which the quality of medical care would deteriorate, in which quality would become secondary to cost.

He said American medicine is the best in the world, medical education unsurpassed, and the qualifications of U. S. physicians unmatched.

"Ours is a dynamic system of health care—and it works," he said. "The very fact that we now have 16½ million Americans 65 years of age and older proves that it works.

"Yet, this same system of medical care is now under attack. At a moment when American medicine is pre-eminent throughout the world, it is proposed that we adopt the very systems under which one European nation after another has lost its former leadership in medical science.

"The staggering costs of such plans, the administrative problems they create—let

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these considerations be secondary," he said. "The important thing is to see, at close range, the disruption of the doctor-patient relationship; the delays in admission to hospitals; the time wasted in the over-crowded offices of doctors; the regimentation of medical practice; the effect of the program on medical research; the availability of medical facilities and personnel—in other words, medicine in action on a government-run, assembly-line basis."

Dr. Larson said also:

1. Congress is being asked to plunge into a compulsory government-operated program of health care for certain of the country's elderly without knowing what even the first-year cost will be—whether $1 billion or $4 billion—and without any clear idea of the extent of the problem it seeks to solve.

2. The bill under consideration would give a single government official the power to "become the nation's czar of hospital care."

3. Contrary to statements of supporters of the measure that physicians' services are not included in the program, more than 50,000 doctors would be directly affected by regulations and controls exercised by government over operations and administration of hospitals.

4. Enactment of the program would "lower the quality of medical care available to the older people of the United States" because "it would introduce into our system of freely practiced medicine elements of compulsion, regulation and control" by government.

5. The Administration proposal is unnecessary in light of the true economic status of the aged and because of the spectacular rise of voluntary, private health insurance coupled with passage by Congress of the Kerr-Mills Medical Aid for the Aged Law last year and the existence of other public and private programs of aid to the needy.

6. Health care at the expense of the working people would be provided for millions who are financially able to pay for their own care.

7. The legislation "proposes that we distrust the brains and capacities of today's Americans," because "it suggests that the aged—as an entire group—are not capable of looking after their own affairs and providing for their own needs."

8. Increasing costs of the program could impose such a financial strain on social security that the entire system could be jeopardized.

9. The Administration's bill is just as objectionable as the five similar health care proposals rejected by Congress since 1942.

10. The bill would violate "American ideals of independence, self-sufficiency, and personal responsibility" by establishing a system in which medical aid would be provided not on the basis of need but on the basis of age.

Dr. Larson described estimates of the cost of the Administration program as "confusing."

The A. M. A. president reminded committee members that HEW Secretary Abraham Ribicoff had told them that "a closer study" had revealed it would be necessary to increase the taxable wage base from the present $4,800 to $5,200, rather than the $5,000 fixed in the bill when it was introduced.

He also pointed out that HEW originally had said nursing home services during the first year of operation of the Administration scheme would cost $9 million.

But in May, Dr. Larson said, HEW officials reported the figure as "unrealistically low" and lifted it to "somewhere between $25 million and $25.5 million."

"Obviously this estimate is something less than precise," Dr. Larson said.

The A. M. A. president said that supporters of the Administration proposal have built their case on five false premises: 1) that the sociologic problems of older people can be solved through legislation; (2) that most, if not all, of the aged are in poor health; (3) that most, if not all, of the aged are verging on bankruptcy; (4) that the problem of the aged in financing their health costs will get worse before it gets better, and (5) that voluntary health insurance and prepayment plans, private effort, and existing law will not do the job that needs doing.

1961
TRANSACTIONS
OF THE
AUXILIARY TO THE MEDICAL SOCIETY
OF THE STATE OF NORTH CAROLINA

THIRTY-EIGHTH ANNUAL MEETING
HELD AT
ASHEVILLE, NORTH CAROLINA
MAY 7-9, 1961

President, Mrs. George T. Noel, Kannapolis
Recording Secretary, Mrs. Daniel S. Currie, Parkton
Treasurer, Mrs. Ralph Deaton, Jr., Greensboro

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Transactions
1961

AUXILIARY TO THE MEDICAL SOCIETY
of the
STATE OF NORTH CAROLINA

Memorial Service, Sunday, May 7, 1961

The Memorial Service of the Auxiliary to the Medical Society of the State of North Carolina was held Sunday, May 7, 1961, at 8 P.M., together with that of the Medical Society, in the Gold Room, Battery Park Hotel, Asheville. The Invocation was given by the Reverend Charles D. Davis, Mars Hill College, Mars Hill, N. C. The Roll Call of the sixty-six deceased physicians of the Society was given by Charles H. Pugh, M.D., Chairman of the Committee on Necrology, Gastonia, N. C., with appropriate words in memoriam.

Mrs. William P. Richardson, Chairman of the Memorials Committee of the Auxliary to the Medical Society, presented the list of sixteen deceased Auxiliary members*, with words of tribute.

A choral presentation was given by the Mars Hill College Choir, under the direction of Thomas J. Cole. The program included "Let All Ye Sons and Daughters Sing", "Look Unto Me", and "Praise Ye the Lord".

Dr. Pugh introduced Dr. Ben F. Royal, Morehead City, Committee member, and then turned the program over to Dr. C. T. Wilkinson, Wake Forest, who was substituting for Dr. Amos N. Johnson. Dr. Wilkinson in turn introduced the Reverend Charles D. Davis, Mars Hill College, who delivered the Memorial address. This was followed by a Choral Postlude and Benediction.

***Deceased Auxiliary Members, 1960-61**

Mrs. McTyeire Gallant Anders, Gastonia
Mrs. Elbert Carl Anderson, Wilmington
Mrs. George Gaylord Gilbert, Asheville
Mrs. Samuel Crawford Gillespie, Asheville
Mrs. William Ray Griffin, Sr., Asheville
Mrs. Claude Milnot Jones, Greenville
Mrs. Clarence Manteo Lentz, Albemarle
Mrs. Roy Hardin Moore, Canton
Mrs. Charles Collins Orr, Asheville
Mrs. James Lowry Pressly, Statesville
Mrs. Edward McQueen Salley, Hendersonville
Mrs. Louis Cotten Skinner, Greenville
Mrs. William Ivey Taylor, Sr., Burgaw
Mrs. Fred Gwyn Woodruff, High Point
Mrs. George Elwood Gorganus, Jacksonville
Mrs. Wirt Wilsey Smith, Durham

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

Finance Committee Meeting—May 8, 1961

Present: President, President-Elect, First Vice-President, Treasurer, Recording Secretary

The Finance Committee met for coffee and rolls in the President's Suite, at 9:15 A.M., May 8, 1961, with Mrs. W. Ralph Deaton, Treasurer, presiding. Mrs. Deaton presented the Financial Statement, with explanations. After discussion, it was decided that the Committee would accept this statement, with three recommendations to the Executive Committee:

1) That we approve the necessary excess expenditure for the **Auxiliary News**;

2) That, for accounting and investing purposes, $3.21 be taken from Miscellaneous to make the Yoder Bed Endowment Fund a round sum, $2650.00;

3) That all money over $500.00 in the Sanatoria Bed Fund be turned over to the Mental Health Research Endowment Fund; this, in view of the fact that the Yoder Endowment Fund is now completed.

Mrs. Deaton then presented two alternate budgets for 1961-62; this was because of the recommendation (with regard to dues) to be voted on at the meeting of the House of Delegates; both these were approved by the Finance Committee, the one to be presented depending on the vote in the House of Delegates.

There being no further business, the meeting was adjourned.

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

Executive Committee Meeting, May 8, 1961

Present: Mrcs. Joseph M. Hitch, Mrs. George T. Noel, Mrs. Robert L. Garrard, Mrs. W. Ralph Deaton, Jr., Mrs. Paul P. McCain, Mrs. Robert D. Croom, Jr., Mrs. Harry D. Riddle.

The Executive Committee met in the Beau Nash Room, The Manor, at 10:00 A.M., with Mrs. Joseph M. Hitch presiding. Recommendations from the Finance Committee (see minutes Finance Meeting) were approved, with the Parliamentarian, Mrs. Robert D. Croom, Jr., stating that it was not necessary for these to be passed by the House of Delegates, since the Finance Committee and Executive Committee had passed upon them, and it was thus necessary only for the President to make a statement to that effect.

Mrs. Hitch brought a suggestion from Watauga County that there be some state-wide Auxiliary project, such as "bulbs" or "brooms". This was discussed and referred to the President-Elect for further action if so desired.

Mrs. Robert D. Croom, Jr., made the suggestion that the Auxiliary introduce a physical fitness program, to be undertaken on a state-wide level, with the hope that North Carolina might perhaps be the state to lead the national Auxiliary to such a program. This was enthusiastically received, and specific recommendations will be given at the Fall Board Meeting.

With no further business the meeting was adjourned.

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

Board of Directors Annual Meeting
May 8, 1961, 11:00 A.M.

The 38th Annual Meeting of the Board of Directors of the Auxiliary to the Medical Society of the State of North Carolina was called to order by the President, Mrs. Joseph M. Hitch, in the Beau Nash Room, The Manor, Asheville.

Mrs. Candler A. Willis, before giving the Invocation, asked that we have a special thought for Dr. and Mrs. Roscoe D. McMillan, in the recent loss of Dr. McMillan's son. Following this, the motion was made, seconded, and passed, to dispense with the Roll Call and reading of the Minutes, in the interest of time.

Convention Chairman, Mrs. Lewis Lunsford, Jr., made announcements concerning the Wild Flower Tour, and rides to the luncheons.

Mrs. Hitch introduced guests, Mrs. W. Walton Hubbard, Southern Regional Vice-President, Womans' Auxiliary to the American Medical Association, and Mrs. Henry E. Steadman, Treasurer, Auxiliary to the Southern Medical Association.

Supplementary reports, bringing the work of the Auxiliary up to date, were distributed.

Mrs. Paul P. McCain, Chairman of Past Presidents, introduced the past presidents, of whom four others were present as follows:

Mrs. A. Byron Holmes, Fairmont
Mrs. Robert D. Croom, Jr., Maxton
Mrs. Donnie M. Royal, Salemburg
Mrs. Robert L. Garrard, Greensboro.

First Vice-President, Mrs. Robert L. Garrard, introduced the three District Councilors present, Mrs. Phil Barringer, 7th District, Mrs. William M. Long, 9th District, Mrs. Candler A. Willis, 10th District, and two substitutes, Mrs. John Robertson, 5th District, and Mrs. Elmer A. Lodmell, 8th District.

In the absence of the Second Vice-President, Mrs. James F. Reinhardt, Mrs. Hitch introduced the Sanatoria Bed Chairmen present—Mrs. John L. McCain, Cooper Bed, and Mrs. Eugene C. Clayton, Stevens Bed. She also recognized Mrs. Phil L. Barringer, substitute for Mrs. John W. Ormand, Chairman of Student Loan Fund. Mrs. Barringer called attention to the supplement to the Annual Report, concerning the Student Loan Fund.

At this point, Mrs. Paul P. McCain asked permission to convey the thanks of the North Carolina Sanatoria Board of Directors (of which she is a member) for all the work of the Medical Auxiliary with the Sanatoria beds.

Bulletin Chairman, Mrs. Thomas E. Fitz, announced that there were 155 subscriptions this year, and that the smaller auxiliaries had done better than the large ones. She emphasized the necessity for every county officer and chairman to subscribe to this publication, and asked for suggestions about getting more subscriptions.

Civil Defense Chairman, Mrs. Amos N. Johnson, stated that the number of participating members was larger than actually shown by the Annual Report.

Community Health Chairman, Mrs. John C. Reece, had no addition to the Annual Report, but mentioned that the combination of Community Health and Service Committees will make annual reporting much easier.

Doctors' Day Chairman, Mrs. Carroll C. Lupton, stated that she had one other report, in addition to those in the Annual Report. Mrs. Amos N. Johnson commented on what auxiliaries in Florida, for instance, were doing for Doctors' Day, and Mrs. Robert L. Garrard added that Doctors' Day observance, originally a project of Southern Medical Auxiliary, was now being taken up in other states.

Memorials Chairman, Mrs. William P. Richardson, reported that she had read the list of sixteen deceased Auxiliary members at the annual Memorial Service (see Minutes of Memorial Service), and asked the group to stand in silent prayer in tribute to these members.

Mental Health Chairman, Mrs. Andrew M. Lang, announced that every district had made a report of some activities, but she had no addition to the Annual Report.

Nominations Chairman, Mrs. Robert L. Garrard, was present, and stated that she would keep her report for the next day, at the meeting of the House of Delegates.

Safety Chairman, Mrs. Lacy J. Parsons, Jr., was absent, but Mrs. R. Vernon Jeter asked to announce that Martin-Washington-Tyrell, with an Auxiliary of seven members, had won a safety award from the North Carolina Rural Safety Council in the past year.

Convention Chairman, Mrs. Lewis Lunsford, Jr., reminded the group that tickets to the President-elect luncheon and the general luncheon were still to be picked up; also that rides would be available to the Biltmore Country Club for the luncheon, and for the Wild Flower tour later.

The date for the Fall Board Meeting and Workshop was announced by Mrs. George T. Noel, President-elect, as Tuesday, August 29, 1961, at the Cabarrus Country Club, Kannapolis.

Representatives to other organizations were recognized: Mrs. John C. Reece, Advisory Committee to the Medical Society on Rural Health and Education; Mrs. Donnie M. Royal, Councilor to the Auxiliary to the Southern Medical Association; Mrs. Calmers R. Carr, Florence Crittenton Home, who stated that the Director of the Home had offered to give programs for any county auxiliaries who might be interested. In answer to a question, Mrs. Carr also said that the Florence Crittenton Home would be delighted to have clothes of any sort, maternity, baby, or otherwise. The representative to the N. C. Council of Women's Organizations, Mrs. John K. Robertson, announced that the Leadership Training Workshop would be held July 17-20, in Chapel Hill.

Mrs. Hitch called attention to the fact that our Auxiliary, for the first time, this year became a member of the N. C. Rural Safety Council. She recognized Mrs. Annette Boutwell, Rural Health Consultant for the Medical Society.

Mrs. Chalmers R. Carr, Charlotte, asked to call the attention of the group to the series of articles on facilities—and lack of—for mentally ill children in N. C., in the **Charlotte Observer**, by Don Seaver, and said that these could be obtained in full for the asking, from the **Charlotte Observer.**

The Recording Secretary was asked to read the amended section of the By-Laws, combining Section 5 and 6 (see page 4, Minutes of Delegates Meeting, May 10, 1960), concerning the Nominating Committee. Mrs. Hitch then asked for nominations for the Nominating Committee, and the following slate was unanimously chosen:

Mrs. Joseph M. Hitch (immediate Past President, automatically the Chairman)
Mrs. W. Ralph Deaton, Jr., 8th District
Mrs. Phil L. Barringer, 7th District
Mrs. Paul P. McCain, 4th District
Mrs. Amos N. Johnson, 3rd District

and two alternates—

Mrs. Lewis Lunsford, Jr., 10th District
Mrs. Bruce B. Blackmon, 5th District.

UNFINISHED BUSINESS

The Recording Secretary was asked to read three recommendations from the Finance and Executive Committees as follows:

1) That we approved the necessary excess expenditure for the **Auxiliary News**;

2) That, for accounting and investing purposes, $3.21 be taken from the Miscellaneous to make the Yoder Bed Endowment Fund a round sum, $2650.00;

3) That all money over $500.00 in the Sanatoria Bed Fund be turned over to the Mental Health Research Endowment Fund; this, in view of the fact that the Yoder Endowment Fund is now completed.

These recommendations were approved by the Board.

NEW BUSINESS

Mrs. Robert D. Croom, Jr., was recognized, to present a new idea for consideration, that we introduce into our acitivities a physical fitness program. She pointed out the findings in the last few years, that while roughly 57% of European youth can pass a simple physical fitness test, only about 8% of American youth can. Commenting that this is a program worthy of concentrated attention, that it has been recognized both on a state and national level as being of great importance, and that North Carolina is the only state with an executive director for such a program, she called for this to be incorporated as a program of our Auxiliary for the next year. She went even further in saying that it is to be hoped that perhaps N. C. might be the state to lead the national Auxiliary into a concentrated endeavor in this direction. This suggestion had been presented to, and approved by, the Executive Committee earlier. The Board was most approving of this idea, and it will be brought up again in th form of specific recommendations at the Fall Board Meeting and Workshop.

Mrs. Hitch called attention to the Auxiliary exhibits in the Gold Room, The Manor.

Mrs. Lewis Lunsford, Jr., suggested that a telegram be sent to Dr. and Mrs. Roscoe D. McMillan, long beloved by the N. C. Medical Auxiliary, expressing sympathy in the recent death of Dr. McMillan's son; also a letter to Mrs. Charles F. Strosnider, past President, who is ill. The Secretary was so directed.

With no further business, the meeting was adjourned.

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

House of Delegates Annual Meeting—May 9, 1961

The 38th Annual Meeting of the House of Delegates of the Auxiliary to the Medical Society of the State of North Carolina met in Great Hall, The Manor, Asheville, on Tuesday May 9, 1961, at 9:00 A.M. The meeting was called to order by the President, Mrs. Joseph M. Hitch, and the invocation was given by Mrs. William P. Richardson. A motion was made, seconded, and passed, to dispense with the Roll Call and the reading of the Minutes.

Mrs. Hitch at this time pointed out that this particular meeting was not one of reports, but of recognition. She introduced guests present: Mrs. W. Walton Hubbard, Southern Regional Vice-President, Woman's Auxiliary to the American Medical Association, and Miss Agnes Campbell, President N. C. League for Nursing, who brought greetings from her organization, and spoke briefly about the work of the League.

REPORT OF OFFICERS

Mr. Hitch then gave her President's Report (attached), obviously considered by the House as a most commendable resume of a valuable year's work; the motion to accept this report was made by Mrs. Carroll C. Lupton, seconded by Mrs. A. Byron Holmes, and enthusiastically passed by the group.

Chairman of Past Presidents, Mrs. Paul P. McCain, introduced the other six Past Presidents who were present:

Mrs. A. Byron Holmes, Fairmont
Mrs. Harry L. Johnson, Elkin
Mrs. Gilbert M. Billings, Morganton
Mrs. Robert D. Croom, Jr., Maxton
Mrs. Donnie M. Royal, Salemburg
Mrs. Robert L. Garrard, Greensboro.

Mrs. McCain also suggested at this time that all delegates, officers, etc., write their first names on their tags.

PRESIDENT-ELECT, Mrs. George T. Noel, was recognized. She stated that the Fall Board Meeting and Workshop would be held at the Cabarrus Country Club, Kannapolis, Tuesday, August 29, 1961.

FIRST VICE-PRESIDENT, Mrs. Robert L. Garrard, introduced District Councilors present, who in turn introduced County Presidents or representatives (see attached program). In the 9th District, Councilor Mrs. William M. Long announced the organization of a new Auxiliary, in Davidson County.

In the absence of the Second Vice-President, Mrs. James F. Reinhardt, Mrs. Hitch introduced the Activities Chairmen (on attached program) present.

Other officers were recognized: Recording Secretary, Mrs. Harry D. Riddle; Parliamentarian, Mrs. Robert D. Croom, Jr.; and Treasurer, Mrs. W. Ralph Deaton, Jr. Mrs. Deaton at this time presented the Financial Statement, with explanations approved by the Board of Directors, and this statement was accepted by the House.

COMMITTEE CHAIRMEN

AMEF Chairman, Mrs. Bruce B. Blackmon, reported the final total of contributions as $2790.15. Mrs. Hitch added that six counties were on record as contributing to the Medical School of the University of N. C., in addition to a contribution from the Past Presidents' Fund.

Memorials Chairman, Mrs. William P. Richardson, repeated her tribute from the Memorial Service of Sunday night, reading the names of the sixteen deceased Auxiliary members, and the group stood for a moment of silent prayer in their honor (see Mem. Service Minutes for list).

Mrs. Lewis Lunsford, Jr., Convention Chairman, reported that there were 44 delegates registered. At this point, Mrs. Hitch expressed the appreciation of the Auxiliary for all the work of the Buncombe County Auxiliary as hostesses for the Convention.

Representatives to other State organizations were recognized. Mrs. Hitch mentioned that Martin-Washington-Tyrell, with an Auxiliary of seven members, had won a safety award from the N. C. Rural Safety Council this year. The N. C. Medical Auxiliary has just become a member of this organization in the past year.

UNFINISHED BUSINESS

The Recording Secretary was asked to read the five recommendations from the Board of Directors Meeting, September 7, 1960:

1) That the First Vice-President become an elected officer in charge of membership and organization, having direct supervision of the work of the Councilors and giving their report at the Annual Meeting. Further, that Article VI, Section 3, and Article V, Section 2, be amended accordingly. The motion was made by Mrs. A. Byron Holmes, seconded by Mrs. Robert L. Garrard, and passed.

2) That the immediate Past President, who becomes a Director, serve as Chairman of Reports for one year, with full responsibility for preparing report forms for the use of committee chairmen, these forms to be prepared and presented to the President for inclusion in the packets at the Fall Board Meeting. Further, that Article VI, Section I, be amended accordingly with the addition of this subsection. Motion was made Mrs. Harry L. Johnson, seconded by Mrs. Paul P. McCain, and passed.

3) That, to avoid duplication, the committees of Community Health and Community Service be combined, this committee to be known as Community Service beginning in May, 1961. Further, that Article XIV, Section 2, be amended accordingly. Motion was made by Mrs. John C. Reece, seconded by Mrs. Carroll C. Lupton, and passed.

4) That, following the policy of the National Auxiliary, a Committee for Rural Health be established to begin its activities in May, 1961. Further, that Article XIV, Section 2, be amended accordingly. Motion was made by Mrs. C. Henry Sikes and seconded by Mrs. William P. Richardson; question was raised why the Committee for Rural Health was being established again, when there used to be one which was done away with. Mrs. Garrard explained that it was true there used to be a Committee on Rural Health, but then it was felt that the term "Rural" might be undesirable to some Auxiliary workers. Later, the trend changed (national), and it was decided that there should again be a Rural Health Committee. After this discussion, the recommendation was passed.

5) That, due to rising costs of printing, clerical personnel, and postage, dues be raised by fifty cents, **becoming $2.50 yearly in 1961-62,** $1.00 to be sent to the National Auxiliary and $1.50 to be used to defray the expenses of the State Auxiliary. (Specific amounts are not mentioned in our By-Laws; therefore, no amendment is necessary.) Motion was made by Mrs. W. Ralph Deaton, Jr., seconded by Mrs. Carroll C. Lupton, and passed.

NEW BUSINESS

The President called upon the Treasurer, Mrs. W. Ralph Deaton, Jr., to present the tentative Annual Budget. After this presentation, Mrs. Deaton moved that the budget be accepted; the motion was seconded by Mrs. Daniel S. Currie, Jr., and was passed unanimously by the House.

The Recording Secretary was asked to read the list of Nominating Committee members for 1961-62. These were as follows:

Mrs. Joseph M. Hitch (Past President automatically becoming Chairman)
Mrs. W. Ralph Deaton, Jr., 8th District
Mrs. Phil L. Barringer, 7th District
Mrs. Paul P. McCain, 4th District
Mrs. Amos N. Johnson, 3rd District;
and two alternates:
Mrs. Lewis Lunsford, Jr., 10th District
Mrs. Bruce B. Blackmon, 5th District

The President announced that the Board of Directors had approved three changes in the budget: 1) the necessary excess expenditure for the **Auxiliary News**; 2) that, for accounting and investment purposes, $3.21 be taken from the Miscellaneous Fund to make the Yoder Bed Endowment Fund a round sum, $2650.00; 3) that all money over $500.00 in the Sanatoria Bed Fund be turned over to the Mental Health Research Endowment Fund; this latter in view of the fact that the Yoder Endowment Fund is now completed.

Mrs. Hitch reminded everyone to be sure to see the Auxiliary exhibits in the Gold Room of the Manor, and pointed out that the point system used to judge the anual awards was detailed on a large poster there. She also announced that the Secretary had been directed to send a telegram to Dr. and Mrs. Roscoe D. McMillan, expressing sympathy in the recent death of Dr. McMillan's son; and also a letter to Mrs. Charles F. Strosnider, Past President, who is ill.

The meeting was then adjourned for a Coca-Cola break before the General Meeting.

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

GENERAL MEETING

May 9, 1961
10:45 A.M.

The 38th Annual General Meeting of the Auxiliary to the Medical Society of the State of North Carolina convened in Great Hall, The Manor, Asheville, and was called to order by the President, Mrs. Joseph M. Hitch. The Invocation was given by Mrs. A. Bryon Holmes, and was followed by the Pledge of Loyalty, with all participating.

A warm welcome was given by Mrs. Michael F. Kelcher, President of Buncombe County Auxiliary, followed by a gracious response by Mrs. Paul E. Simpson, President of Wake County Auxiliary. Mrs. Hitch also expressed her gratitude for the excellent work of preparation that our hostesses had done.

Concerning Your Health and Your Income

THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA SPECIAL GROUP ACCIDENT AND HEALTH PLAN IN EFFECT SINCE 1940

This is our 21st year of service to the Society. It is our aim to continue to lead the field in pr ing Society members with disability protection and claim services as modern as tomorrow.

SPECIAL FEATURES ARE:

1. **Up to a possible 7 years for each sickness (no confinement required).**
2. **Pays up to Lifetime for accident.**
3. **New Maximum limit of $650.00 per month income while disabled.**

All **new** applicants, and those now insured, who are under 55, and in good health, are eligib apply for the new and extensive protection against sickness and accident.

BENEFITS AND RATES AVAILABLE UNDER NEW PLAN

			COST UNTIL AGE 35		COST FOR AGES 35 1	
Accidental Death Coverage	***Dismemberment Loss of Sight, Speech or Hearing**	**Accident and Sickness Benefits**	**Annual Premium**	**Semi-Annual Premium**	**Annual Premium**	**Semi-A Premi**
5,000	5,000 to 10,000	50.00 Weekly	$ 78.00	$ 39.50	$104.00	$ 52
5,000	7,500 to 15,000	75.00 Weekly	114.00	57.50	152.00	76
5,000	10,000 to 20,000	100.00 Weekly	150.00	75.50	200.00	100
5,000	12,500 to 25,000	125.00 Weekly	186.00	93.50	248.00	124
5,000	15,000 to 30,000	150.00 Weekly	222.00	111.50	296.00	148

***Amount payable depends upon the nature of the loss as set forth in the policy.**

OPTIONAL HOSPITAL COVERAGE: Members under age 60 in good health may apply for $2 daily hospital benefit—Premium $20.00 semi-annually or $40.00 annually.

Write, or call us collect (Durham 682-5497) for assistance or information

ALL CLAIMS ARE PAID IMMEDIATELY FROM OUR OFFICE.

Administered by
J. L. CRUMPTON, State Mgr.
Professional Group Disability Division
Box 147, Durham, N. C.
J. Slade Crumpton, Field Representative

UNDERWRITTEN BY THE COMMERCIAL INSURANCE COMPANY OF NEWARK, N. J.
Originator and pioneer in professional group disability plans.

AN IMPORTANT STATEMENT

TO MEMBERS OF THE MEDICAL SOCIETY

HOSPITAL SAVING ASSOCIATION

Your Blue Shield Plan

values the confidence and support of the physicians of North Carolina. Hospital Saving Association has cooperated fully with the Medical Society through a mutual Statement of Understanding entered into several years ago by both parties.

- Since it was nationally approved in 1946, the Association has conformed to all national requirements and cooperated with other Blue Shield Plans throughout the nation.
- The official Blue Shield emblem symbolizes national approval. For the Association, it also represents a cherished asset, one that has been built through untiring efforts, promotional expenditures—and with the understanding and cooperation of the Society itself for the past fifteen years.
- Through its extensive statewide sales organization, the Association continues to make Blue Shield available to North Carolinians. It is proud of its 1960 increase of over 61,000 participants—a gain of 12.78% over 1959.

In 1940, at the request of Dr. I. H. Manning, the House of Delegates permitted Hospital Saving Association to write Surgical and Medical programs. Thus, from the beginning, the Association has operated under the aegis of Medical Society leadership.

In 1946, Hospital Saving was included in the first group of Plans that were given the privilege of using the copyrighted Blue Shield emblem. National approval has been continuous; Hospital Saving has always considered it a sacred trust to protect and build the fine national and local concepts of Blue Shield.

More recently, the Medical Society instructed the Association to provide the DOCTORS PROGRAM of service benefits, which has grown in public acceptance to the point where over 31% of the Association's membership—and over 36% of its professional claims—are now under this program.

®

Dr. Amos N. Johnson, President of the Medical Society, brought official greetings from the Society. He complimented the Auxiliary on the work it had done through the years, both tangible and intangible. He emphasized againthat the greatest problem ahead of us is the fight against a welfare state, and socialized medicine. He pointed out that the medical profession has been becoming more and more fragmented, that it must remain a group of medical doctors, not specialists, in order to be effective in this fight, and asked us to encourage our husbands to participate actively in organized medicine. He introduced two guests he had brought with him—Dr. Julian Price, Chairman of Boar dof Trustees, AMA, and Mr. William Waddell, medical students at Duke University and Past President of SAMA.

Mrs. Hitch thanked Dr. Johnson, and introduced other guests present: Mrs. E. Vincent Askey, wife of the President of the AMA; Miss Agnes Campbell, President, N. C. League for Nursing; Mrs. W. Walton Hubbard, Southern Regional Vice-President, Woman's Auxiliary to AMA; Mrs. Henry E. Steadman, Treasurer, Auxiliary to the Southern Medical Association.

She announced that the Secretary had sent a telegram to Dr. and Mrs. Roscoe D. McMillan expressing sympathy in the recent death of Dr. McMillan's son, and a note to Mrs. Charles F. Strosnider, past President, who is ill.

There followed a musical entertainment by Mrs. John W. Ledbetter and Mr. John Bridges, of Asheville. They sang both duets and solos from "Kismet", "Music Man", "The King and I", and finished with a Victor Herbert duet, "Thine Alone".

Mrs. Paul P. McCain introduced the Past Presidents, of whom six others were present: Mrs. A. Byron Holmes, Fairmont; Mrs. Harry L. Johnson, Elkin; Mrs. Gilbert M. Billings, Morganton, Mrs. Robert D. Croom, Jr., Maxton; Mrs. Donnie M. Royal, Salemburg; Mrs. Robert L. Garrard, Greensboro.

Mrs. Hitch remarked that we have three National board members: Mrs. Robert D. Croom, Jr., member of the Board of Directors; Mrs. Robert L. Garrard, member of the Publications Committee; Mrs. Donnie M. Royal, Southern Regional Chairman of Mental Health.

At this time, Mrs. Hitch introduced for an address, Mrs. W. Walton Hubbard, Southern Regional Vice-President of the Woman's Auxiliary to AMA who emphasized what Dr. Johnson had said earlier—that we must assume responsibilities or someone else will assume them for us. She stressed that what the critics of medicine write and say, **do** have a great effect on our public relations, and that what we say **to everyone** is important. She called particular attention to "Operation Coffee Cup", saying that the aim is 100 letters a week to the Senate and House. She brought out that since 1952, AMEF has invested over one million dollars in medical students; that we have one new Auxiliary in the national organization, Puerto Rico; and with an inspiring finish, asked that we at all times try to make friends for the Auxiliary.

Mrs. Hitch then called upon Mrs. J. W. Roy Norton ,replacing Mrs. Powell G. Fox, to present the annual awards. They were as follows:

AMEF—Shirley Kingsbury Fox Award—Mrs. Powell G. Fox, Raleigh
Winner: Lincoln County—$100.00
First Honorable Mention—Stanly County
Second Honorable Mention—Johnston County

DOCTORS' DAY—Mrs. Harvey C. May, Charlotte
Guilford (Greensboro)—50 members or more—$5.00
Johnston County—less than 50 members—$5.00
Honorable Mention—Alamance-Caswell

FIRST 100% DUES PAID—Mrs. Gilbert M. Billings, Morganton
Winner—Stanly County—$5.00
First Honorable Mention—Union County

HEALTH CAREERS—The Rachel Taylor Award—Mrs. Almon R. Cross, High Point
Forsyth-Stokes—over 30 members—$5.00
Johnston County—less than 30 members—$5.00

MENTAL HEALTH—Mrs. Karl B. Pace, Greenville
Wilson County—$5.00

MENTAL HEALTH RESEARCH ENDOWMENT FUND—Mrs. Robert L. Garrard, Greensboro
Winner—Lincoln County—$10.00
First Honorable Mention—Chowan-Perquimans
Second Honorable Mention—Wake County
Others—Johnston, Robeson, Guilford (Greensboro), Mecklenburg

PRESIDENT'S COUNTY ACHIEVEMENT AWARD—Mrs. Joseph M. Hitch, Raleigh
Winner—Catawba County—$10.00
First Honorable Mention—Gaston County
Second Honorable Mention—Forsyth-Stokes

RESEARCH AND ROMANCE OF MEDICINE—Mrs. Donnie M. Royal, Salemburg
First—Guilford (Greensboro)—$5.00
Second—Gaston County—$5.00

STUDENT LOAN FUND—Mrs. Roscoe D. McMillan and Mrs. B. Watson Roberts, Red Springs and Durham
Winner—Watauga County—$10.00

First Honorable Mention—Union County
Second Honorable Mention—Forsyth-Stokes

YEARBOOK (COUNTY)—Mrs. Baxter S. Troutman, Lenoir
Winner—Mecklenburg—$5.00

YODER ENDOWMENT FUND—Mrs. Robert D. Croom, Jr., Maxton
Winner—Chowan-Perquimans—$5.00
First Hinorable Mention—Columbus County
Second Honorable Mention—Forsyth-Stokes

SCRAPBOOK—judged at convention, no monetary award, merely "recognition"
Winner—Rowan-Davie
Second—Durham-Orange
Third—Forsyth-Stokes

The President then called upon our other special guest, Mrs. Henry E. Steadman, Treasurer, Auxiliary to Southern Medical Association, who spoke briefly. She reminded us that Southern was the originator of Doctors' Day, a project that is now gradually being take up by other sections of the country. She announced that the annual convention would be in Dallas, Texas, on November 6-9, and hoped that many of us could be present.

The President asked for the report of the Nominating Committee, and Mrs. Robert L. Garrard, Chairman, gave it as follows:

President-elect—Mrs. C. Tolbert Wilkinson, Wake Forest
First Vice-President—Mrs. John C. Reece, Morganton
Recording Secretary—Mrs. Daniel S. Currie, Jr., Fayetteville

The floor was opened to nominations, of which there were none. The motion was made by Mrs. A. Byron Holmes to accept the slate as read, seconded by Mrs. Robert L. Garrard, and it was passed, the Secretary being instructed to cast a unanimous ballot for each of the nominees.

Mrs. Paul P. McCain, in her traditional role, then installed the following officers for 1961-62:

President—Mrs. George T. Noel
President-elect—Mrs. C. Tolbert Wilkinson
Recording Secretary—Mrs. Daniel S. Currie, Jr.

Mrs. Hitch, with appropriate words of congratulations, presented the gavel to Mrs. Noel, who then presented the Past President's pin to Mrs. Hitch.

Mrs. Noel, as the new President, made a few inaugural remarks, pledging to continue the work of her predecessors.

Mrs. Lewis Lunsford, Jr., Convention Chairman, announced that the final registration count was 246 Auxiliary members, and 81 guests.

With no further business, the meeting was adjourned.

Mrs. Harry D. Riddle
Recording Secretary

Mrs. Joseph M. Hitch
President
Date: June 1, 1961

Report of Treasurer and Auditors Report

The Audited Report of the Treasurer's records for the year 1960-1961 is submitted herewith, receipts and disbursements having been recorded and transactions made in accordance with the Bylaws.

A membership of 2,276 was attained in 1960-1961 which is only three less than last year. We consider this fortunate because we did not hear from two counties that were supposed to be organized.

The financial picture of the Auxiliary is stable. We did not have to go into the Checking Account Contingency Fund which is represented by share interests of $2,000.00 in the Home Federal Savings and Loan Association, Greensboro, but instead have received 4% interest on this amount this year.

Two United States Savings Bonds for the Stevens Endowment Fund matured in January and were reinvested in United States Treasury Notes that will yield 4% interest. As a result of taking advantage of securing four United States Treasury Notes off the market, for the past two years, the income for the Stevens Endowment Fund has increased by $54.00 yearly.

Contributions to our various projects from the Counties were very generous. The Paul Allison Yoder Endowment Fund has now been completed at $10,150.00. The Mental Health Research Fund is up to $696.92. The Student Loan Fund has a balance of $1,652.90. Two loans were issued last year amounting to $950.00. We are making a second loan of $550.00 next year and will be able to help another new student.

My job as Treasurer continues to be an interesting and pleasurable experience. This year I have used secretarial help when possible which has taken a good deal of the drudge out of the job. I have enjoyed working with the Executive Board and especially with Mrs. J. M. Hitch, our president for the past year. I am delighted that she will continue to be Financial Advisor for the coming year. My thanks go to everyone who worked with the Auxiliary to make this a good financial year.

Mrs. W. Ralph Deaton, Jr.
State Treasurer

REPORT ON EXAMINATION

June 30, 1961

Mrs. W. Ralph Deaton, Jr., Treasurer
The Auxiliary to the Medical Society of the State of North Carolina
Greensboro, North Carolina

Dear Mrs. Deaton:

We have examined the cash receipts and disbursements records of the Auxiliary to the Medical Society of the State of North Carolina for the fiscal year ended June 30, 1961. Our examination was made in accordance with generally accepted auditing standards and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

We herewith present our report which consists of the following statements:

Exhibit A—Statement of Assets and Funds Balances
Exhibit B—Statement of Cash Receipts and Disbursements
Schedule 1—Investments
Schedule 2—Notes Receivable—Student Loans
Schedule 3—Cash Disbursements

In our opinion, the accompanying statements present fairly the assets and funds balances arising from cash transactions of the Society at June 30, 1961, and the cash receipts and disbursements transactions for the year then ended on a basis consistent with that of the preceding year.

Respectfully submitted,

Davenport, Marvin & Caudle
Certified Public Accountants

EXHIBIT A

THE AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

STATEMENT OF ASSETS AND FUNDS BALANCES

June 30, 1961

ASSETS

Current Funds:			
Cash in Bank—Checking Account		$ 1,595.08	
Cash in Bank—Savings Account		2,000.00	
Total Current Funds			$ 3,595.08
Endowment Funds:			
Cash in Bank—Savings Accounts:			
Student Loan Fund	$ 1,707.87		
Mental Health Research Fund	1,308.88		
Paul Yoder Endowment Fund	150.00		
		3,166.75	
Investments (at cost)—Schedule 1		42,000.00	
Notes Receivable—Student Loans—Schedule 2		5,924.97	
Total Endowment Funds			51,091.72
Total Assets			$54,686.80

FUND BALANCES

Current Funds:		
General Expense Fund	$ 507.21	
Secretarial Aid Fund	151.62	
Sanatoria Bed Fund	500.00	
Convention Expense Fund	436.25	
Contingency Fund	2,000.00	
Total Current Funds		$ 3,595.08
Endowment Funds:		
Student Loan Fund	7,632.84	
Mental Health Research Fund	1,308.88	
Paul Yoder Endowment Fund	10,150.00	
Martin L. Stevens Endowment Fund	10,500.00	
George M. Cooper Endowment Fund	10,000.00	
McCain Endowment Fund	11,500.00	
Total Endowment Funds		51,091.72
Total Fund Balances		$54,686.80

EXHIBIT B

THE AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

STATEMENT OF CASH RECEIPTS AND DISBURSEMENTS

For the Fiscal Year Ended June 30, 1961

	Total Current Funds	Current Funds					Student Loan Fund	Mental Health Research Fund	Paul Yoder Endowment Fund
		General Expense	Secretarial Aid	Sanatoria Bed	Convention Expense	Contingency			
Receipts:									
National Dues	$ 2,264.00	$2,264.00	$	$	$	$	$	$	$
State Dues	2,264.00	2,264.00							
Medical Society of the State of North Carolina:									
National Fall Conference	300.00	300.00							
President's Secretarial Aid	300.00		30						
Convention Expenses	500.00				500.00				
Interest on U. S. Treasury Savings Bonds:									
Stevens Endowment Fund	334.50			334.50					
Cooper Endowment Fund	276.00			276.00					
McCain Endowment Fund	317.40			317.40					
Yoder Endowment Fund	225.00			225.00					
Interest on Savings Accounts:									
Contingency Fund	80.00	80.00							
Student Loan Fund							42.95		
Mental Health Research Fund								12.85	
Yoder Endowment Fund									90.24
Interest on Student Loan Receivable							32.47		
Payment on Student Loan Receivable							25.03		
Contributions							1,099.30	594.70	574.25
Sale of "Handbook" and "Histories"	7.50	7.50							
Overpayment of Dues	6.00	6.00							
Transfers From:									
Yoder Endowment Fund	2,697.63			2,697.63					
Student Loan Fund	950.00	950.00							
Sanatoria Bed Fund								601.33	217.40
Total Receipts	10,522.03	5,871.50	300.00	3,850.53	500.00	.00	1,199.75	1,208.88	881.89
Disbursements—Schedule 3	10,197.06	5,753.61	148.38	3,917.93	377.14	.00	950.00	.00	2,697.63
Excess of Receipts Over Disbursements	324.97	117.89	151.62	(67.40)	122.86	.00	249.75	1,208.88	(1,815.74)
Cash on Hand, July 1, 1960	3,270.11	389.32*	.00	567.40*	313.39	2,000.00	1,458.12	100.00	1,965.74
Cash on Hand, June 30, 1961	$ 3,595.08	$ 507.21	$151.62	$ 500.00	$436.25	$2,000.00	$1,707.87	$1,308.88	$ 150.00

*Adjusted for error in the amount of $19.00 in prior year's report.

THE AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA
SCHEDULE OF INVESTMENTS
June 30, 1961

Schedule 1

Kind	Series	Date Issued		Maturity Date		Total	Endowment Fund Martin L. Stevens	George M. Cooper	McCain	Paul Yoder
U. S. Treasury Savings Bonds	G	July	1950	July	1962	$ 2,000.00	$ 2,000.00			
U. S. Treasury Savings Bonds	G	June	1951	June	1963	1,000.00	1,000.00			
U. S. Treasury Notes		July	1959	May	1964	2,000.00	2,000.00			
U. S. Treasury Savings Bonds	K	June	1952	June	1964	1,000.00	1,000.00			
U. S. Treasury Savings Bonds	K	August	1954	August	1966	1,500.00		$ 1,500.00		
U. S. Treasury Savings Bonds	K	September	1954	September	1966	2,500.00			$ 2,500.00	
U. S. Treasury Savings Bonds	K	May	1955	May	1967	1,500.00		1,500.00		
U. S. Treasury Savings Bonds	K	June	1955	June	1967	11,000.00		3,000.00	7,000.00	$ 1,000.00
U. S. Treasury Savings Bonds	K	August	1955	August	1967	6,000.00		1,000.00	2,000.00	
U. S. Treasury Savings Bonds	K	September	1955	September	1967	500.00	500.00			
U. S. Treasury Savings Bonds	H	January	1958	January	1968	1,000.00				1,000.00
U. S. Treasury Savings Bonds	H	March	1958	March	1968	500.00				500.00
U. S. Treasury Savings Bonds	K	May	1956	May	1968	2,000.00				2,000.00
U. S. Treasury Savings Bonds	K	June	1956	June	1968	1,500.00				1,500.00
U. S. Treasury Savings Bonds	K	April	1957	April	1969	1,500.00	1,000.00			500.00
U. S. Treasury Savings Bonds	H	February	1959	February	1969	1,000.00				1,000.00
U. S. Treasury Savings Bonds	H	September	1959	September	1969	1,000.00	1,000.00			
U. S. Treasury Notes		October	1960	October	1969	2,000.00	2,000.00			
U. S. Treasury Savings Bonds	H	June	1961	June	1971	2,500.00				2,500.00
						$42,000.00	$10,500.00	$10,000.00	$11,500.00	$10,000.00

Schedule 2

STUDENT LOANS
June 31, 1961

Name	Amount
Jerome Miles Schachter	$ 474.97
Norris A. Biggerstaff	500.00
Thomas A. Readling	1,000.00
Ronnie Lee Stanley	1,000.00
John Edwin Drew	1,000.00
Robert H. Fleming	500.00
Dick Redmond Lavender	1,000.00
Michel Larome Barringer	150.00
Total	$5,924.97

Note—Student loans are payable three years after date of note and bear interest @ 3% per annum on unpaid balance therefrom. Following notes due at June 30, 1961:

Name	Dated	Amount
Jerome Miles Schachter	10-15-55	$ 474.97
Norris A. Biggerstaff	1-30-57	500.00
Total		$ 974.97

THE AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

SCHEDULE OF CASH DISBURSEMENTS

For the Fiscal Year Ended June 30, 1961

		Current Funds							
	Total Current Funds	General Expense	Secretarial Aid	Sanatoria Bed	Convention Expense	Contingency	Student Loan Fund	Mental Health Research Fund	Paul Yoder Endowment Fund
National Dues	$ 2,277.00	$2,277.00	$	$	$	$	$	$	$
Officers and Chairmen Expenses:									
President	700.51	552.13	148.38						
President-Elect	250.00	250.00							
Vice-Presidents and Secretary	56.04	56.04							
Treasurer	220.81	220.81							
Councilors	71.15	71.15							
Committee Chairmen	188.12	188.12							
Auxiliary News	463.71	463.71							
Science Fair	50.00	50.00							
Refund of Overpayment of Dues	5.00	5.00							
Past President's Pins	27.50	27.50							
Student Loan Grants	950.00	950.00							
Safety Deposit Box Rental	5.50	5.50							
Professional Services—Auditing	100.00	100.00							
Treasurers' Fidelity Bond	50.00	50.00							
Printing and Supplies	486.65	486.65							
Purchase of U. S. Treasury Savings Bond (Yoder Endowment Fund)	2,500.00			2,500.00					
Upkeep of Sanatoria Beds:									
McCain (N. C. Sanatorium)	121.20			121.20					
Cooper (Eastern N. C. Sanatorium)	72.00			72.00					
Stevens (Western N. C. Sanatorium)	219.00			219.00					
Yoder (Graveley Sanatorium)	178.20			178.20					
Refund to U. S. Treasury (Interest on Savings Bond)	8.80			8.80					
Convention Expenses (other than luncheon meal)	377.14				377.14				
Transfers to:									
Mental Health Research Fund	601.33			601.33					
Yoder Endowment Fund	217.40			217.40					
Sanatoria Bed Fund									2,697.63
General Expense Fund							950.00		
Total Disbursements	$10,197.06	$5,753.61	$148.38	$3,917.93	$377.14	$ .00	$950.00	$ .00	$2,697.63

ROSTER OF MEMBERS
1961-1962

HONORARY MEMBERS

Holmes, Mrs. Andrew Byron
112 Church Street .... Fairmont
Judd, Mrs. E. Clarence
2108 Woodland Ave. .... Raleigh
Knight, Mrs. William Pinkney
720 Summit Ave. .... Greensboro
McCain, Mrs. Paul Pressly
Route 1, Box 31 .... Wilson
Taylor, Mrs. Frederick R.
1113 Johnson St. .... High Point

LIFE MEMBERS

Britt, Mrs. James Norment
209 E. 10th St. .... Lumberton
Eldridge, Mrs. Charles Patterson
1621 St. Mary's St. .... Raleigh
Freeman, Mrs. Jere David
527 Forest Hills Dr. .... Wilmington
Johnson, Mrs. George W.
1803 Chestnut St. .... Wilmington
Murray, Mrs. Robert Lebby
Box 216 .... Raeford
Thomas, Mrs. Charles Darwin...... Black Mountain
Yoder, Mrs. Paul A.
1919 Robin Hood Rd. .... Winston-Salem

MEMBERS

Abbott, Mrs. Robert West
Cherry Hospital .... Goldsboro
Abernathy, Mrs. Henry .... Hickory
Abernethy, Mrs. Paul McBee
510 Country Club Dr. .... Burlington
Adair, Mrs. William Edward, Jr.
502 East G St. .... Erwin
Adams, Mrs. Carlisle
1500 Meadowood Lane .... Charlotte
Adams, Mrs. Carlton Noble
2930 Windsor Rd. .... Winston-Salem
Adams, Mrs. Charles H.
Box D .... Grover
Adams, Mrs. Charles Patrick
1907 Forest Hill Dr. .... Greenville
Adams, Mrs. Harley Stewart
432 Carolina Circle .... Winston-Salem
Adams, Mrs. Simeon Huey
1413 Lineberger Ave. .... Gastonia
Ader, Mrs. Ottis Ladeau .... Walkertown
Aderholt, Mrs. Marcus Lafayette, Jr.
1013 Rotary Dr. .... High Point
Adickes, Mrs. Henning Frederick, Jr.
3832 Selwyn Ave. .... Charlotte
Adkins, Mrs. Trogler Francis
2810 Dogwood Rd. .... Durham
Agner, Mrs. Marshall Edward
Box 157 .... Cherryville
Agner, Mrs. Roy Augusta, Jr.
400 Merritt Ave. .... Salisbury
Albergotti, Mrs. Julian S., Jr.
412 Livingston Dr. .... Charlotte
Alderman, Mrs. Allison Mondonville, Jr.
1311 Westfield Ave. .... Raleigh
Alderman, Mrs. Edward H.
Drawer P .... Four Oaks
Alexander, Mrs. Eben, Jr.
1941 Georgia Ave. .... Winston-Salem
Alexander, Mrs. George Thomas
Erwin Heights .... Thomasville
Alexander, Mrs. Henry C., Jr.
221 Chase St. .... Charlotte
Alexander, Mrs. James Moses
255 Colville Rd. .... Charlotte
Alexander, Mrs. James Porter
1910 Beverly Dr. .... Charlotte
Alexander, Mrs. Joseph Black
N. Elm St. .... Lumberton
Alexander, Mrs. Lawrence M.
308 Green St. .... Sanford
Alexander, Mrs. Sydenham B.
511 Dogwood Dr. .... Chapel Hill
Alexander, Mrs. William McKinley
1110 Fourth Ave., West .... Hendersonville
Allen, Mrs. Charles Insley, Sr. .... Wadesboro
Allen, Mrs. George Calvin
206 E. 17th St. .... Lumberton
Allen, Mrs. James Norman
109 Maxwell Rd. .... Chapel Hill
Allen, Mrs. John O. Henry
201 Broad St. .... Marion
Allen, Mrs. Joseph J. .... Warrenton
Allen, Mrs. LeRoy
1603 Ridge Rd. .... Raleigh
Allen, Mrs. William
Knollwood .... Southern Pines
Allgood, Mrs. John William, Jr.
105 Knollwood Dr. .... Greensboro
Alsup, Mrs. William Byrn, Jr.
261 Westview Dr. .... Winston-Salem
Altany, Mrs. Franklin Edward
822 Longbow Rd. .... Charlotte
Alyea, Mrs. Edwin Pascal
3102 Devon Rd., Hope Valley .... Durham
Ames, Mrs. Richard Haight
2316 Princess Ann St. .... Greensboro
Anders, Mrs. McTyeire Gallant
416 W. 5th Ave. .... Gastonia
Anderson, Mrs. John Bascom
294 Vanderbilt Rd. .... Asheville
Anderson, Mrs. Norman LaRue
33 Forest Road .... Asheville
Anderson, Mrs. William Banks
502 E. Forest Hills Blvd. .... Durham
Andrew, Mrs. John Montgomery
3½ N. Main St. .... Lexington
Andrew, Mrs. Lacy Allen, Jr.
2839 Reynolds Rd. .... Winston-Salem
Andrews, Mrs. Bob Barcus
503 W. 31st .... Lumberton
Andrews, Mrs. Leon Polk
2217 Winterlocken Rd. .... Fayetteville
Andrews, Mrs. Robert Jackson
1130 S. Live Oak Parkway .... Wilmington

Andrews, Mrs. Vernon Liles ....Mt. Gilead
Angel, Mrs. Edgar ....Franklin
Anlyan, Mrs. William George
1124 Woodburn Rd. ....Durham
Anthony, Mrs. Luther Leslie
1210 Jones St. ....Gastonia
Anthony, Mrs. William Augustus
1203 Belvedere Ave. ....Gastonia
Antonakos, Mrs. Theodore ....Danbury
Arey, Mrs. John Vincent
89 Caldwell Dr. ....Concord
Armistead, Mrs. Drury Branch
1603 E. 6th St. ....Greenville
Armstrong, Mrs. Beverly Weller
1 Armstrong Drive ....Charlotte
Armstrong, Mrs. Charles Wallace
629 Mitchell Ave. ....Salisbury
Arney, Mrs. William Charles
W. Park Drive ....Morganton
Arnold, Mrs. Jesse Hoyt, Jr.
709 W. Highland Ave. ....Kinston
Arnold, Mrs. Ralph A.
911 Urban Ave. ....Durham
Arrendell, Mrs. Cad Walder, Jr.
500 Merwick Circle ....Charlotte
Arthur, Mrs. Robert
308 Hinsdale Ave. ....Fayetteville
Arthur, Mrs. Robert Key, Jr.
405 Rolling Rd. ....High Point
Ashe, Mrs. John Rainey, Jr.
203 Grandview Dr. ....Concord
Ashford, Mrs. Charles Hall
605 Pollock St. ....New Bern
Atkins, Mrs. Stanley Sisco
7 N. Dogwood Rd. ....Asheville
Ausband, Mrs. John Rufus
817 Shoreland Rd. ....Winston-Salem
Ausherman, Mrs. Howard Milton
233 Fenton Place ....Charlotte
Austin, Mrs. Frederick DeCosta, Jr.
650 Colville Rd. ....Charlotte
Averett, Mrs. Leland Stanley, Jr.
1506 Whitehall ....High Point
Avery, Mrs. Edward Stanley
1824 Meadowbrook Dr. ....Winston-Salem
Aycock, Mrs. Edwin Burtis
Longmeadow Rd. ....Greenville
Aycock, Mrs. James Bernice
110 Maehill Dr. ....Lenoir
Aycock, Mrs. William Glenn
E. Graham St. ....Mebane
Ayers, Mrs. James Salisbury
Finch St. ....Clinton

Bagby, Mrs. Bathurst Browne, Jr.
17 Howland Rd. ....Asheville
Baggett, Mrs. Joseph Woodrow
365 Valley Rd. ....Fayetteville
Bailey, Mrs. Clarence Whitfield
512 Shady Circle Dr. ....Rocky Mount
Bailey, Mrs. Joseph Peden..................Hendersonville
Bailey, Mrs. Lloyd Whitfield
3208 Hawthorne Rd. ....Rocky Mount
Bailey, Mrs. Mercer H.
Winslow Acres ....Elizabeth City
Bailey, Mrs. Robert Carl
330 Scenic Dr. ....Concord
Baker, Mrs. Barnwell Rhett
31 Buena Vista Rd. ....Asheville
Baker, Mrs. Herbert Marvin ....Faith
Baker, Mrs. Horace Mitchell, Sr.
703 N. Elm St. ....Lumberton
Baker, Mrs. Horace Mitchell, Jr.
1901 N. Elm St. ....Lumberton
Baker, Mrs. Larry Duane
3116 Gardner Park Dr. ....Gastonia
Baker, Mrs. Lenox Dial
3106 Cornwall Rd., Hope Valley ....Durham
Baker, Mrs. Roger D.
303 Swift Ave. ....Durham
Baker, Mrs. Thomas Williams
2029 Queens Rd. ....Charlotte
Baldwin, Mrs. William Edwin, Jr.
Wilmington Rd. ....Whiteville
Ballenger, Mrs. Claude Newton
750 Pee Dee Ave. ....Albemarle
Ballew, Mrs. James Robert
901 Lake Boone Trail ....Raleigh
Balsley, Mrs. Robert Eugene
825 Crescent Drive ....Reidsville
Baluss, Mrs. John William, Jr.
2315 Westdale Dr. ....Fayetteville
Bandy, Mrs. William Henry
Dogwood Hills ....Newton
Banner, Mrs. Charles Whitlock
808 N. Elm St. ....Greensboro
Barden, Mrs. Graham Arthur, Jr.
412 Johnson St. ....New Bern
Barefoot, Mrs. Graham Ballard
120 Forest Hills Dr. ....Wilmington
Barefoot, Mrs. Julius J., Sr.
609 Middle St. ....New Bern
Barefoot, Mrs. Julius Jackson, Jr.
Old Cherry Point Rd. ....New Bern
Barefoot, Mrs. Sherwood Washington
3107 Madison Ave. ....Greensboro
Barefoot, Mrs. William Frederick
Chadbourn Rd. ....Whiteville
Barker, Mrs. Christopher Sylvanus
711 Broad St. ....New Bern
Barker, Mrs. Julian
1223 Ashcraft Lane ....Charlotte
Barnes, Mrs. Frank Edward, Jr.
513 Church St. ....Smithfield
Barnes, Mrs. Henry Eugene, Jr.
528 First Ave., N. W. ....Hickory
Barnes, Mrs. M. Russell, Jr.
401 Woodland Dr. ....Jacksonville
Barnhardt, Mrs. Albert Earl, Box 662, Kannapolis
Barnhill, Mrs. Otha Allen, Box 505, Elizabethtown
Barrett, Mrs. John Milton
805 James St. ....Greenville
Barrick, Mrs. Harry, Jr.
1900 Highland Place ....Raleigh
Barrier, Mrs. Cecil Lee
Route 3 ....Lawndale

Barrier, Mrs. Henry Webster
1500 Central Dr. .... Concord
Barringer, Mrs. Archie Lipe
Box 278 .... Mt. Pleasant
Barringer, Mrs. Phil Lewis
Forest Hills .... Monroe
Barringer, Mrs. Thad Jones
Rt. 6, Leadmine Rd. .... Raleigh
Barron, Mrs. Archie A.
2251 Selwyn Ave., Apt. 4 .... Charlotte
Barron, Mrs. John Isaac
508 Riverside Dr. .... Morganton
Barry, Mrs. William
505 Cole Ave. Ext. .... Raeford
Barry, Mrs. William Francis, Jr.
1022 Gloria Ave. .... Durham
Bartels, Mrs. Kenneth Garber
312 Regal .... Hendersonville
Bartlett, Mrs. Stephen Russell, Jr.
208 N. Longmeadow Rr. .... Greenville
Bates, Mrs. Harold Bascom
1007 Sherwood Dr. .... Burlington
Batten, Mrs. Hubert Elmore
301 Fairfield Rd. .... Fayetteville
Batten, Mrs. Woodrow
402 Church St. .... Smithfield
Bauer, Mrs. Harold Alfred
Box 338 .... Wrightsville Beach
Baylin, Mrs. George Jay
2535 Wrightwood Ave. .... Durham
Baynes, Mrs. Ralph H. .... Hurdle Mills
Beale, Mrs. Seth McPherson
Box 588 .... Elkin
Beall, Mrs. Lawrence Lincoln
1850 North Elm St. .... Greensboro
Bear, Mrs. Sigmond Aaron
1415 S. Live Oak Parkway .... Wilmington
Beavers, Mrs. Charles Lee
1110 Sunset Dr. .... Greensboro
Beavers, Mrs. James Wallace
2206 W. Market St. .... Greensboro
Beavers, Mrs. William Olive
Route 1 .... McLeansville
Beck, Mrs. J. Montgomery
Route 7 .... Burlington
Beddingfield, Mrs. Edgar Theodore, Jr.
.... Stantonsburg
Belcher, Mrs. Cecil Cullen
28 Hilltop .... Asheville
Belk, Mrs. George Washington
403 W. 6th Ave. .... Gastonia
Bell, Mrs. George Erick, Sr.
1505 W. Nash St. .... Wilson
Bell, Mrs. Ira Eugene
508 6th St., N. W. .... Hickory
Bell, Mrs. Orville Earl
829 Sycamore St. .... Rocky Mount
Bell, Mrs. Spencer Alexander
Box 33 .... Hamptonville
Bell, Mrs. William Harrison, Jr.
P. O. Box 1298 .... New Bern
Bellamy, Mrs. Robert Hartlee
Greenway Ave. .... Wilmington

Benbow, Mrs. Edgar Vernon
1411 Reynolda Rd. .... Winston-Salem
Benbow, Mrs. Edward Perry, Jr.
3809 Friendly Road .... Greensboro
Bender, Mrs. John Joseph .... Red Springs
Bender, Mrs. John Robert
1166 S. Hawthorne Rd. .... Winston-Salem
Bennett, Mrs. Ernest Claxton
Box 295 .... Elizabethtown
Bennett, Mrs. Harron Kent .... Archdale
Bennett, Mrs. Hugh Hammond, Jr.
441 Circle Dr. .... Burlington
Bennett, Mrs. John Northwood
c/o Wilkes General Hospital North Wilkesboro
Bennett, Mrs. Paul, Jr.
109 S. Andrews .... Goldsboro
Bensen, Mrs. Vladimir Basil
Blue Ridge Road .... Raleigh
Benson, Mrs. John Fisher
710 Gatewood .... High Point
Benson, Mrs. Norman Oliver
203 E. 19th St. .... Lumberton
Benson, Mrs. Walter Russell
Coker Drive .... Chapel Hill
Benton, Mrs. George Ruffin, Jr.
207 S. Pineview Ave. .... Goldsboro
Berkeley, Mrs. Alfred Rives, Jr.
541 Hempstead Place .... Charlotte
Berkeley, Mrs. Scott Bruce, Jr.
1108 Evergreen Ave. .... Goldsboro
Berkeley, Mrs. William Thomas, Jr.
1870 Queens Rd., W. .... Charlotte
Berry, Mrs. Francis Xavier
1208 Colonial Ave. .... Greensboro
Berryhill, Mrs. Walter Reece
Box 866, Upper Laurel Hill .... Chapel Hill
Bertling, Mrs. Marion Henry
2312 Princess Ann St. .... Greensboro
Best, Mrs. Deleon Edward
1504 E. Mulberry St. .... Goldsboro
Best, Mrs. William Ross
1510 Sherwood Dr. .... Burlington
Bethea, Mrs. William Thad .... Fair Bluff
Bethel, Mrs. Millard B.
303 Weaver Rd. .... Chapel Hill
Betts, Mrs. Wilmer Conrad
3615 Alleghany .... Raleigh
Biggs, Mrs. Dennis Walter, Jr.
205 West 22 St. .... Lumberton
Biggs, Mrs. John Irvin
2201 Elm St. .... Lumberton
Billings, Mrs. Gilbert M.
122 Powe St. .... Morganton
Bingham, Mrs. Robert Knox
105 Hardin St. .... Boone
Bingham, Mrs. W. L.
Long Street .... Lexington
Bitting, Mrs. Numa Duncan
34 Oak Dr. .... Durham
Bittinger, Mrs. Charles Lewis
734 Pinewood Circle .... Mooresville
Bittinger, Mrs. Samuel Moffett
Blue Ridge Rd. .... Black Mountain

Bivens, Mrs. Edward Shirley
601 East St. .... Albemarle
Bizzell, Mrs. James W.
Overbrook Drive .... Goldsboro
Bizzell, Mrs. Marcus Edward
500 E. Walnut St. .... Goldsboro
Black, Mrs. George William
1566 Queens Rd., W. .... Charlotte
Black, Mrs. John Riley, Jr.
212 Jefferson St. .... Whiteville
Black, Mrs. Paul Adrian Lawrence
2732 Park Ave. .... Wilmington
Blackmon, Mrs. Bruce Bernard .... Buie's Creek
Blackwelder, Mrs. Verne Hamilton
323 S. Mulberry St. .... Lenoir
Blair, Mrs. Andrew B.
1220 Queens Rd., W. .... Charlotte
Blair, Mrs. George Walker, Jr.
460 Parkview Dr. .... Burlington
Blair, Mrs. James Samuel
1116 Cumberland Ave. .... Gastonia
Blair, Mrs. Mott Parks .... Marshville
Blake, Mrs. Damon
645 Kingsbury Circle .... Winston-Salem
Blanchard, Mrs. George Caswell
1701 Brandon Rd. .... Charlotte
Blanchard, Mrs. Irvin T.
1409 Riverside Ave. .... Elizabeth City
Bland, Mrs. Delmar Earl
289 Canterbury Trail .... Winston-Salem
Bland, Mrs. William Herbert
401 West .... Cary
Bliss, Mrs. Forrest Edgar
Drawer G .... Lawndale
Block, Mrs. Milton Edgar
522 S. State St. .... Lexington
Blowe, Mrs. Ralph Boyd, Sr.
800 Washington Ave. .... Weldon
Blue, Mrs. John Frederick
522 Brinn Drive .... Sanford
Boatwright, Mrs. Robert Stuart .... Hazelwood
Boggs, Mrs. Lawrence Kennedy
2208 Wellesley Ave. .... Charlotte
Bolin, Mrs. Grover Cleveland, Jr.
Crescent Drive .... Smithfield
Bolin, Mrs. Lewis Bryant
111 Will St. .... Mt. Airy
Bolon, Mrs. Charles Gordon
4733 Wendover Lane .... Charlotte
Bolt, Mrs. Conway Anderson
Box 368 .... Marshville
Bolus, Mrs. Michael
2220 Wheeler Rd. .... Raleigh
Bond, Mrs. Edward Griffith
102 Pembroke Circle .... Edenton
Bond, Mrs. John Lawrence
Wilkes General Hospital .... North Wilkesboro
Bond, Mrs. John Pennington
1806 Fairfield Dr. .... Gastonia
Bond, Mrs. Vernard Franklin, Jr.
340 Buckingham Rd. .... Winston-Salem
Bonner, Mrs. John Bryan Havens
1100 Riverside Ave. .... Elizabeth City

Bonner, Mrs. Merle Dumont
203 Kimberly Dr. .... Greensboro
Boone, Mrs. William Waldo
1001 Gloria Ave. .... Durham
Borden, Mrs. Richard Winstead
1600 E. Elm St. .... Goldsboro
Boren, Mrs. Richard Benjamin III
813 N. Bridge St. .... Elkin
Bos, Mrs. John Fremont
1574 Clayton Dr. .... Charlotte
Bourgeois, Mrs. Michael
1017 Norwood Ave. .... Durham
Bower, Mrs. Joseph Shelton
1100 N. Queens St. .... Kinston
Bowles, Mrs. Francis Norman
1400 Shepherd St. .... Durham
Boyce, Mrs. Oren Douglas
Boyceleyn Rd .... Gastonia
Boyce, Mrs. William Henry
939 N. Straford Rd. .... Winston-Salem
Boyd, Mrs. Basil Manley, Jr.
1816 Maryland Ave. .... Charlotte
Boyd, Mrs. Joseph Alston, Jr.
409 Chestnut St. .... Henderson
Boyes, Mrs. James Gordon, Jr.
104 Forest Hills Dr. .... Wilmington
Boyette, Mrs. Ben Robert, Jr.
1508 E. Palm St. .... Goldsboro
Bradford, Mrs. George Edwin
4444 Roslyn Rd. .... Winston-Salem
Bradford, Mrs. Wallace Brown
256 Hempstead Pl. .... Charlotte
Bradford, Mrs. Williamson Ziegler
310 Colville Rd. .... Charlotte
Bradish, Mrs. Robert F.
1712 Raeford Rd. .... Fayetteville
Bradley, Mrs. Harold John
105 W. Brentwood .... Greensboro
Bradley, Mrs. John David
5 Ravenna Dr. .... Asheville
Bradshaw, Mrs. Howard Holt
2837 Reynolds Rd. .... Winston-Salem
Bradsher, Mrs. James Donald
Box 168 .... Roxboro
Brady, Mrs. Charles Eldon
Carthage Rd. .... Robbins
Brady, Mrs. Walter Morris .... Morehead City
Branaman, Mrs. Guy Hewitt, Jr.
915 Williamson Dr. .... Raleigh
Brandon, Mrs. Henry Allen .... Yadkinville
Brantley, Mrs. Coleman
1803 Wright Ave. .... Greensboro
Brantley, Mrs. Julian Thweatt
714 Summit Ave. .... Greensboro
Bream, Mrs. Charles Anthony
Button Road .... Chapel Hill
Breeden, Mrs. William Henry
1524 Morganton Rd. .... Fayetteville
Brenizer, Mrs. Addison Gorgas, Jr.
1301 Providence Rd. .... Charlotte
Bressler, Mrs. Bernard
2700 Circle Dr. .... Durham
Brewer, Mrs. James Street .... Roseboro

Brian, Mrs. Earl Winfrey
2111 White Oak Rd. ... Raleigh
Brice, Mrs. George Wilson, Jr.
3961 Arbor Way ... Charlotte
Bridger, Mrs. Dewey Herbert ... Bladenboro
Bridges, Mrs. Dwight Thomas ... Lattimore
Briggs, Mrs. Henry Harrison, Jr.
323 Vanderbilt Rd. ... Asheville
Brigman, Mrs. Paul Hamer
1005 College Dr. ... High Point
Brinkhous, Mrs. Kenneth Merle
524 Dogwood Ave. ... Chapel Hill
Brinn, Mrs. Thomas Preston
106 Front St. ... Hertford
Britt, Mrs. Benjamin E.
1009 Stancil Dr. ... Raleigh
Britt, Mrs. Tilman Carlisle, Jr.
130 Rawley Ave. ... Mt. Airy
Britt, Mrs. Walter S.
Veterans Hospital ... Fayetteville
Brittain, Mrs. Lowell Ellis ... Huntersville
Brockmann, Mrs. Harry Lyndon
912 Fairway Dr. ... High Point
Bronnenberg, Mrs. Neel
421 5th St., N. W. ... Hickory
Brooks, Mrs. Ernest Bruce
2853 Bitting Rd. ... Winston-Salem
Brooks, Mrs. Frederick Philips
Greenville Blvd. ... Greenville
Brooks, Mrs. Ralph Elbert
1303 Rainey St. ... Burlington
Brooks, Mrs. William Lester, Jr.
2110 Queens Rd., W. ... Charlotte
Broughton, Mrs. Arthur Calvin, Jr.
3008 Eton Rd. ... Raleigh
Broun, Mrs. Matthew Singleton
606 Roanoke Ave. ... Roanoke Rapids
Brouse, Mrs. Ivan Edwin
Box 1198 ... Wilmington
Brown, Mrs. Charles William
227 Fenton Place ... Charlotte
Brown, Mrs. Frank Reid
1103 Country Club Dr. ... Greensboro
Brown, Mrs. George Wallace, Jr. ... Waynesville
Brown, Mrs. Gerald Joseph ... Westfield
Brown, Mrs. Ivan W., Jr.
1709 Vista Dr. ... Durham
Brown, Mrs. James Stevens, Sr.
Hendersonville Inn ... Hendersonville
Brown, Mrs. James Walter, Jr.
33 Grandview Dr. ... Concord
Brown, Mrs. Kermit English
Chunns Cove Rd. ... Asheville
Bruce, Mrs. James Crawford
2902 Dublin ... Greensboro
Brunson, Mrs. Edward Porcher
804 Pee Dee Ave. ... Albemarle
Bruton, Mrs. Charles Wilson ... Troy
Bryan, Mrs. Thomas R., Jr.
Finley Park ... North Wilkesboro
Buffaloe, Mrs. William Joseph
Fairview Road ... Raleigh

Bugg, Mrs. Charles Paulett
320 W. Drewry Lane ... Raleigh
Bugg, Mrs. Everett I., Jr.
1544 Hermitage Ct. ... Durham
Buie, Mrs. Roderick Mark, Sr.
119 Kensington Rd. ... Greensboro
Buie, Mrs. Roderick Mark, Jr.
112 Elgin Place ... Greensboro
Bullard, Mrs. George Minson
Forest Lake ... Mebane
Bullard, Mrs. Hoke Vogler
1502 Grove St. ... Wilson
Bullock, Mrs. Duncan Douglas, Sr. ... Rowland
Bulluck, Mrs. Ernest S.
519 Market St. ... Wilmington
Bumgarner, Mrs. John Reed
2101 Mimosa Dr. ... Greensboro
Bunce, Mrs. Paul Leslie
Route 3 ... Chapel Hill
Bundy, Mrs. James Bizzell
2631 Mirror Lake Dr. ... Fayetteville
Bundy, Mrs. William Lumsden
Finley Park ... North Wilkesboro
Bunn, Mrs. David Glenn
Maple St. ... Whiteville
Bunn, Mrs. Richard Wilmot
411 Plymouth Ave. ... Winston-Salem
Burdette, Mrs. Fred McPherson, Jr.
Box 398 ... Southport
Burleson, Mrs. Robert Joe
36 Elk Mountain Scenic Hwy. ... Asheville
Burnett, Mrs. Thomas J. M.
4756 Stafford Circle ... Charlotte
Burns, Mrs. Stanley Sherman, Jr.
2312 Pembroke Ave. ... Charlotte
Burwell, Mrs. John Cole, Jr.
110 Homewood Dr. ... Greensboro
Busby, Mrs. George Francis
Confederate Ave. ... Salisbury
Busby, Mrs. Julian
P. O. Box 56 ... Kannapolis
Busby, Mrs. Trent
530 Confederate Ave. ... Salisbury
Busse, Mrs. Ewald W.
1423 Woodburn Rd. ... Durham
Butler, Mrs. Radford Norman
810 Clovelly Rd. ... Winston-Salem
Butler, Mrs. Raymond Kenneth ... Waynesville
Byerly, Mrs. Frederick Lee
2000 Robin Hood Rd. ... Winston-Salem
Byerly, Mrs. James Hampton
620 Carr St. ... Sanford
Byerly, Mrs. Wesley Grimes, Sr.
211 Highland Ave. ... Lenoir
Byerly, Mrs. Wesley Grimes, Jr.
362 8th St., N. W. ... Hickory
Byrd, Mrs. Charles William
409 S. Orange Ave. ... Dunn
Byrd, Mrs. William Carey
Broughton Hospital ... Morganton
Byrnes, Mrs. Thomas Henderson
919 Mt. Vernon Ave. ... Charlotte

Byrum, Mrs. Clifford Conwell
2616 Wells Ave. ....Raleigh

Cain, Mrs. Frank Coral, Jr.
1519 Pinola Ave. ....Gastonia
Caldwell, Mrs. Eston Robert, Jr.
116 N. Race St. ....Statesville
Caldwell, Mrs. Jesse Burgoyne
1307 Park Lane ....Gastonia
Caldwell, Mrs. Lawrence McClure
406 S. College Ave. ....Newton
Caldwell, Mrs. Robert M.
224 S. Main St. ....Mt. Airy
Caldwell, Mrs. Robert Sims
520 2nd St., N. W. ....Hickory
Callison, Mrs. William Joseph
10 Blackwood Rd. ....Asheville
Cameron, Mrs. George
307 Westview Drive ....Fayetteville
Cameron, Mrs. Joseph Harold
1217 Crescent Ave. ....Gastonia
Camp, Mrs. Edward Hays ....Waynesville
Campbell, Mrs. Frank Highsmith
1400 Lakeview Dr. ________Fayetteville
Campbell, Mrs. James Melvin
2115 Yost Ave. ....Salisbury
Campbell, Mrs. Joseph Lester
306 Kincaid Ave. ....Wilson
Campbell, Mrs. Paul Curtis, Jr.
2215 Meadow Wood Rd. ....Fayetteville
Carpenter, Mrs. Harry M.
743 Austin Lane ....Winston-Salem
Carr, Mrs. Chalmers Rankin
1715 Queens Rd. ....Charlotte
Carr, Mrs. Edward Sleight
3210 Forsyth ....Greensboro
Carrington, Mrs. George Lunsford
139 Piedmont Way ....Burlington
Carroll, Mrs. Charles Fisher
263 Grandview Dr. ....Concord
Carroll, Mrs. Fountain Williams ....Hookerton
Carroll, Mrs. Francis Murray ....Chadbourn
Carter, Mrs. Francis Bayard
2111 Myrtle Dr. ....Durham
Carter, Mrs. Needham Battle
226 Timberlane Road ....Rocky Mount
Carter, Mrs. Numa Richardson, Jr.
435 Leander St. ....Shelby
Casstevens, Mrs. John Claude
130 Pine Valley Cl. ....Winston-Salem
Cates, Mrs. Banks Raleigh, Jr.
2833 Sunset Dr. ....Charlotte
Caughran, Mrs. John H.
4400 Halstead Dr. ....Charlotte
Causey, Mrs. Andrew Jackson
210 Valley Stream Rd. ....Statesville
Caveness, Mrs. Zebulon Marvin
1804 Hillsboro St. ....Raleigh
Caviness, Mrs. Verne Strudwick
913 Vance St. ....Raleigh
Cayer, Mrs. David
2754 Robin Hood Rd. ....Winston-Salem

Cecil, Mrs. Richard C.
2314 Gunston Court ....Fayetteville
Cekada, Mrs. Emil Bogomir
915 Green St. ....Durham
Cernugel, Mrs. Albert Peter ....Chadbourn
Chambers, Mrs. R. T.
400 Randolph St. ....Thomasville
Chambers, Mrs. Robert Edward
313 Ruby Lane ....Gastonia
Chamblee, Mrs. John Sigma
607 E. Church St. ....Nashville
Chandler, Mrs. Edgar Ted
1029-16th Ave. Circle N.W. ....Hickory
Chandler, Mrs. Weldon Porter
Box 458 ....Weaverville
Chapin, Mrs. John Harmon ....Benson
Chapman, Mrs. Charles Granger
6134 Deveron Dr. ....Charlotte
Chapman, Mrs. Edwin James
264 Lakeshore Dr. ....Asheville
Chapman, Mrs. Jesse Pugh, Jr.
81 Sheridan Rd. ....Asheville
Charlton, Mrs. John David
911 Magnolia St. ....Greensboro
Chastain, Mrs. Loren Lee ....Cherryville
Cheek, Mrs. John Merritt, Jr.
1025 Sycamore St. ....Durham
Cheek, Mrs. Kenneth Maurice
402 E. Farriss ....High Point
Chester, Mrs. Pinkney Jones
Pine Grove Rd. ....Southern Pines
Chidester, Mrs. Augustus Benjamin
Haywood Forest ....Hendersonville
Chiles, Mrs. Noah Hampton
Heathcliffe Road ....High Point
Choate, Mrs. Allyn Blythe
1901 Providence Rd. ....Charlotte
Christian, Mrs. Bernie Joseph
2906 Dellwood Dr. ....Greensboro
Chrysler, Mrs. Charles Otis
110 Lanier Ave. ....Charlotte
Citron, Mrs. David Sanford
2100 Cumberland Ave. ....Charlotte
Clapp, Mrs. Hubert Lee
Eastwood Ave. ....Swannanoa
Clark, Mrs. Badie Travis
607 Raleigh Road ....Wilson
Clark, Mrs. DeWitt Duncan, Box 725 ....Clarkton
Clark, Mrs. Douglas Hendon
207 W. 26th St. ....Lumberton
Clark, Mrs. Harold Stevens
9 Lakewood Dr. ....Asheville
Clark, Mrs. Henry Toole, Jr.
Box 1370 ....Chapel Hill
Clarke, Mrs. Len Gordon
606 Fieldcrest Rd. ....Draper
Clarke, Mrs. William Lowe, Jr.
401 7th Ave. Pl., N. W. ....Hickory
Clary, Mrs. William Thomas
507 Chancery Pl. ....Greensboro
Clay, Mrs. Thomas Barger, Jr.
300 N. Third Ave. ....Mayodan

Clayton, Mrs. Eugene Cook
17 St. Charles Pl. ....Asheville
Clinton. Mrs. Roland Smith
1305 Fairfield Dr. ....Gastonia
Clippinger, Mrs. Frank W.
1302 Oakland Ave. ....Durham
Cloninger, Mrs. Charles Edgar ....Conover
Cloninger, Mrs. Giles Lathern
301 Dogwood Lane ....Hamlet
Cloninger, Mrs. Kenneth Lee
Westlake Hills ....Newton
Clutts, Mrs. George Robert
227 N. Park Dr. ....Greensboro
Cobey, Mrs. William Gray
527 Clement Ave. ....Charlotte
Cochcroft, Mrs. Roy Leicester
217 W. Washington Ave. ....Bessemer City
Cochran. Mrs. James R.
730 Richmond ....Laurinburg
Cochran, Mrs. John L., Jr.
413 N. Elm St. ....Asheboro
Cochrane, Mrs. Fred Richard, Jr.
1614 Maryland Ave. ....Charlotte
Codington, Mrs. John Bonnell
422 Forest Hill Dr. ....Wilmington
Coffee, Mrs. Archie Thomas, Jr.
3319 Sharon Rd. ....Charlotte
Coffman, Mrs. Selby
Longmeadow Rd. ....Greenville
Cogdell, Mrs. David Melvin
2827 Skye Dr. ....Fayetteville
Coggeshall, Mrs. Allen Bancroft
109 Beverly Pl. ....Greensboro
Cohen, Mrs. Sanford Irwin
1100 Woodburn Rd. ....Durham
Coker, Mrs. Robert Ervin, Jr.
810 Christopher Rd. ....Chapel Hill
Cole, Mrs. Herman Alfonse
211 E. Blanche ....Clayton
Cole, Mrs. Robert Hickman
1537 Coventry Rd. ....Charlotte
Cole, Mrs. Walter Francis
201 E. Avondale ....Greensboro
Coleman, Mrs. Lester Livingston
428 Sixth St., N. W. ....Hickory
Coley, Mrs. Elwood Brogden
602 W. 31st St. ....Lumberton
Collett, Mrs. James Rountree
312 W. Union St. ....Morganton
Combs, Mrs. Fielding
438 Carolina Circle ....Winston-Salem
Combs, Mrs. Joseph John
2125 White Oak Rd. ....Raleigh
Compton, Mrs. John Wallace
608 S. Ileander Ave. ....Goldsboro
Cook, Mrs. Henry Lilly, Jr.
Irving Park Manor ....Greensboro
Cook, Mrs. Joseph Lindsay
202 Nutbush Rd. ....Greensboro
Cook, Mrs. William Eugene
115 S. Churchill Dr. ....Fayetteville
Cooke, Mrs. Hershall Marcus
Route 1, Box 227 ....Boone
Cooke, Mrs. Quinton Edwin
212 E. High St. ....Murfreesboro
Cooley, Mrs. Samuel Studdiford
221 New Bern Ave. ....Black Mountain
Cooper, Mrs. Albert Derwin
1006 Dacian Ave. ....Durham
Cooper, Mrs. Frank Benton
1129 Emerald St. ....Salisbury
Cooper, Mrs. George Marion
411 Marlowe Road ....Raleigh
Coppedge, Mrs. Thomas Oliver, Jr.
4067 Abingdon Rd. ....Charlotte
Coppridge, Mrs. James Alston
2020 Wilson St. ....Durham
Coppridge, Mrs. William Maurice
1024 W. Forest Hills Blvd. ....Durham
Corbett, Mrs. James Patrick
30 Water ....Swansboro
Corbin, Mrs. George Wesley, Jr. ....Rolesville
Cordell, Mrs. Alfred Robert
963 Kenleigh Circle ....Winston-Salem
Cornwell, Mrs. Abner Milton
825 S. Aspen St. ....Lincolnton
Corpening, Mrs. Joseph Durham
228 Rutherford St. ....Salisbury
Corpening, Mrs. Oscar J. ....Granite Falls
Corpening, Mrs. William Nye ....Granite Falls
Correll, Mrs. Earl Eugene
1603 Eastwood Drive ....Kannapolis
Cosgrove, Mrs. Kenneth Edward
306 Laurel Dr. ....Hendersonville
Costner, Mrs. Walter Vance
501 N. Cedar St. ....Lincolnton
Coughlin, Mrs. Joyce Desmond
150 Cherokee Rd. ....Asheville
Council, Mrs. Albert Barbee
Von Ruck St. ....Spray
Couturier, Mrs. Maurice George, Sr. ....Reidsville
Covington, Mrs. Alpheus M.
Skyland Terrace ....Rockingham
Covington, Mrs. Furman Payne
2 Salem St. ....Thomasville
Covington, Mrs. James Madison, Sr. ....Wadesboro
Covington, Mrs. James Madison, Jr.
507 Morven Road ....Wadesboro
Covington, Mrs. John Malloy Clayton
324 Jackson St. ....Roanoke Rapids
Covington, Mrs. Martin Cade
2107 Woodland Ave. ....Sanford
Cox, Mrs. Alexander McNeil
325 Market St. ....Madison
Cox, Mrs. Samuel Clements
8 E. Bayshore Blvd. ....Jacksonville
Cox, Mrs. William Foscue
2722 Reynolds Rd. ....Winston-Salem
Cozart, Mrs. Benjamin Franklin
Box 1289 ....Reidsville
Cozart, Mrs. Wiley Holt
Box 327 ....Fuquay Springs
Cozart, Mrs. Wiley S.
333 S. Main ....Fuquay Springs
Craddock, Mrs. John Goodwin
1501 Anderson St. ....Wilson

Craig, Mrs. Robert Lawrence
382 Montford Ave. ......Asheville
Craig, Mrs. William Kenneth ......Enfield
Cramblett, Mrs. Henry S.
Wake Forest Faculty Apts. ......Winston-Salem
Crandell, Mrs. Daniel LeRoy
755 Pine Valley Rd. ......Winston-Salem
Crane, Mrs. George William, Jr.
2618 Augusta Dr. ......Durham
Cranford, Mrs. H. P.
23 W. Third St. ......Lexington
Craven, Mrs. Frederick Thorns
29 Ravine Ave. ......Concord
Crawford, Mrs. Robert Orr, Jr.
P. O. Box 483 ......Claremont
Crawford, Mrs. William Jennings
1500 E. Ash St. ......Goldsboro
Creadick, Mrs. Robert Nowell
1200 Anderson St. ......Durham
Creech, Mrs. Lemuel Underwood
220 Edgedale Dr. ......High Point
Creed, Mrs. George Otis
Johns Rd. ......Laurinburg
Crescenzo, Mrs. Victor M.
Belmont Drive ......Reidsville
Crisp, Mrs. Sellers Mark
1201 E. 5th St. ......Greenville
Crissman, Mrs. Clinton S.
Chapel Hill Road ......Graham
Cronland, Mrs. Murphy Allen
226 W. Pine ......Lincolnton
Croom, Mrs. Arthur Bascom
1102 Greenway Dr. ......High Point
Croom, Mrs. Robert DeVane, Jr. ......Maxton
Crosby, Mrs. James Foster
5015 Park Road ......Charlotte
Crosby, Mrs. Lewis Pearce
1210 S. Park Dr. ......Reidsville
Cross, Mrs. Almon Rufus
414 Hillcrest Dr. ......High Point
Cross, Mrs. Robert Vandervoort
920 Fairway Dr. ......High Point
Crouch, Mrs. Auley McRae, Sr.
520 Dock St. ......Wilmington
Crouch, Mrs. Auley McRae, Jr.
1419 S. Live Oak Parkway ......Wilmington
Crouch, Mrs. Walter Lee
1211 S. Live Oak Parkway ......Wilmington
Crow, Mrs. Samuel Leslie
12 N. Kensington Rd. ......Asheville
Crowell, Mrs. James Allen
1529 E. Morehead St. ......Charlotte
Crowell, Mrs. Lester Avant, Jr.
413 S. Aspen St. ......Lincolnton
Crumpler, Mrs. James Fulton
1409 West Haven Blvd. ......Rocky Mount
Crumpler, Mrs. Paul
401 Lafayette St. ......Clinton
Crumpler, Mrs. Warren Harding
N. Johnson St. ......Mt. Olive
Crutchfield, Mrs. Andrew Jackson
300 Plymouth Ave. ......Winston-Salem
Cubberley, Mrs. Charles Lamb, Jr.
505 Lafayette Dr. ......Wilson
Culbreth, Mrs. George Gordon
2228 Queens Rd., E. ......Charlotte
Currie, Mrs. Daniel Smith, Jr.
302 Churchill Dr. ......Fayetteville
Curry, Mrs. Clayton Smith
2701 Bucknell Ave. ......Charlotte
Curtis, Mrs. Thomas E.
Sherwood Forest, Route 2 ......Chapel Hill
Cushman, Mrs. Robert G.
5th Ave. Pl., N. E. ......Hickory
Cutchin, Mrs. Joseph Henry, Sr.
Box 202 ......Whitakers
Cutchin, Mrs. Joseph Henry, Jr. ......Sherrill's Ford
Cutri, Mrs. Joseph John
Graylyn Court ......Winston-Salem

Dale, Mrs. Frederick Payne
503 Rhodes Ave. ......Kinston
Dalton, Mrs. Horace Milton
1705 Cambridge Dr. ......Kinston
Dame, Mrs. Robert L.
214 Ridgeway Ave. ......Statesville
Dameron, Mrs. Thomas Barker, Jr.
414 Scotland St. ......Raleigh
Daniel, Mrs. Crowell Turner, Jr.
330 Pinecrest Dr. ......Fayetteville
Daniel, Mrs. Thomas Brantley
3231 Sussex Rd. ......Raleigh
Daniel, Mrs. Thomas Manning
524 S. Fourth St. ......Smithfield
Daniel, Mrs. Walter Eugene
2115 Roswell Ave. ......Charlotte
Daniels, Mrs. Ralph L.
307 E. Front St. ......New Bern
Daniels, Mrs. Robert Edward
23 Vance Crescent ......West Asheville
Daughtridge, Mrs. Arthur Lee
501 Shady Circle Dr. ......Rocky Mount
Davant, Mrs. Charles
Chestnut Drive ......Blowing Rock
Davenport, Mrs. Carlton Alderman
207 Front St. ......Hertford
Davenport, Mrs. Clifton
309 Third Ave. ......Raeford
Davidson, Mrs. Alan
Old Cherry Point Rd. ......New Bern
Davidson, Mrs. James Hubert
2200 Sprunt St. ......Durham
Davis, Mrs. Courtland Harwell, Jr.
921 Goodwood Rd. ......Winston-Salem
Davis, Mrs. Daniel Whitaker
603 Camden Rd. ......Wadesboro
Davis, Mrs. David A.
Kings Mill Road ......Chapel Hill
Davis, Mrs. Jack Beason ......Waynesville
Davis, Mrs. James Evans
7 Beverly Drive ......Durham
Davis, Mrs. James Matheson ......Wadesboro
Davis, Mrs. Jefferson ......Dallas
Davis, Mrs. John Preston
2425 Queen St. ......Winston-Salem

Davis, Mrs. Joseph Franklin
Box 6291 Summit Station ....Greensboro
Davis, Mrs. Junius Weeks, Jr.
Trent Shores ....New Bern
Davis, Mrs. Philip Bibb
807 Florham Ave. ....High Point
Davis, Mrs. Richard Boyd
122 S. Green St. ....Greensboro
Davis, Mrs. Rufus Jackson
Lakewood ....Cramerton
Davis, Mrs. Wayne Edward
321 Avalon Road ....Winston-Salem
Davis, Mrs. William Hersey, Jr.
723 N. Stratford Rd. ....Winston-Salem
Dawson, Mrs. James Nelson..................Acme-Delco
Dayton, Mrs. John Thomas
2427 Wensley Dr. ....Charlotte
Deaton, Mrs. Paul McNeely
581 Greenway Dr. ....Statesville
Deaton, Mrs. Paul P.
Broughton Hospital ....Morganton
Deaton, Mrs. William Ralph, Jr.
101 Elgin Place ....Greensboro
DeCamp, Mrs. Allen Ledyard
1830 Cassamia Place ....Charlotte
Deeds, Mrs. Charles Ross
Haywood Road ....Hendersonville
Dees, Mrs. Ralph Erastus
1505 Fairmont Street ....Greensboro
DeWolfe, Mrs. Phillip William
Box 106 ....Leaksville
Dick, Mrs. Frederick William
354 Bost St. ....Statesville
Dick, Mrs. Macdonald
3005 Norwich, Hope Valley ....Durham
Dickerson, Mrs. Andrew Jackson........Waynesville
Dickie, Mrs. James William
3003 Wayne Dr. ....Wilmington
Dickson, Mrs. Brice Templeton, Jr.
1436 Fern Forest Drive ....Gastonia
Dickson, Mrs. Malcolm Shields
1903 Woodland Ave. ....Burlington
Dillard, Mrs. Sam Booker
1309 Biltmore Dr. ....Charlotte
Disosway, Dr. Lula M.
1621 Spencer Ave. ....New Bern
Dixon, Mrs. George Grady
503 Snow Hill St. ....Ayden
Dixon, Mrs. Philip Lafayette, Jr.
1 Bayshore Blvd., E. ....Jacksonville
Dobson, Mrs. Richard L.
14 Brandon Rd. ....Chapel Hill
Doerr, Mrs. J. C.
2614 Grant Ave. ....Raleigh
Donald, Mrs. William Blanton, Jr.
603 Rockspring Rd. ....High Point
Donner, Mrs. Paul Gartrell
2910 Abingdon Rd. ....Charlotte
Dorenbusch, Mrs. Alfred A.
2734 Hampton Ave. ....Charlotte
Dorman, Mrs. Bruce Hugh
Rt. 3, Box 163 ....Wilmington
Douglas, Mrs. John Munroe
400 Ferncliff ....Charlotte
Dovenmuehle, Mrs. Robert Henry
3527 Hamstead Court ....Durham
Downs, Mrs. Kenneth Ray
4112 Barmettler Dr. ....Charlotte
Downs, Mrs. Posey Edgar, Jr.
101 Placid Place ....Charlotte
Doyle, Mrs. Owen William
906 Dover Rd. ....Greensboro
Drake, Mrs. Benjamin Michael
1310 Jackson Rd. ....Gastonia
Drake, Mrs. David Ewing
2616 Bennington Rd. ....Fayetteville
Drummond, Mrs. Charles Stitt
2928 Windsor Rd. ....Winston-Salem
Duckett, Mrs. Charles Howard ....Canton
Duckett, Mrs. Virgil Howard ....Canton
Dudley, Mrs. Council C., Jr. ....Jonesville
Duffy, Mrs. Charles
1506 Lucerne Way ....New Bern
Dugger, Mrs. Gordon S.
5 Maxwell Rd. ....Chapel Hill
Dula, Mrs. Frederick Mast
214 Hibriten St. ....Lenoir
Dunlap, Mrs. Jack
6th Street ....Lumberton
Dunn, Mrs. Richard Barry
1014 N. Elm St. ....Greensboro
Dunning, Mrs. Everett Jackson
2501 Danbury St. ....Charlotte
Durham, Mrs. Carey Winston
209 W. Ridgeway Dr. ....Greensboro
Durham, Mrs. Thomas Garrison
502 Crescent Hill ....Kings Mountain
Dyer, Mrs. David Patterson
Rt. 1, Box 18 ....Waynesville

Eagle, Mrs. James Carr
418 Carolina Ave. ....Spencer
Early, Mrs. Ira Gordon
2510 Bitting Rd. ....Winston-Salem
Easom, Mrs. Herman Franklin
508 Mt. Vernon Dr. ....Wilson
Eastwood, Mrs. Frederick Thomas
2708 Lakeview Dr. ....Raleigh
Eckbert, Mrs. William Fox
Southwood, Route 2 ....Gastonia
Eckerson, Mrs. Charles ....Troy
Eddinger, Mrs. Charles Frederick
Box 45, ...Spencer
Eddins, Mrs. George Edgar, Jr.
Norwood Rd. ....Albemarle
Edgerton, Mrs. Glenn Soulders
325 Cherokee Place ....Charlotte
Edwards, Mrs. Charles Daniel
418 E. 12th St. ....Washington
Edwards, Mrs. James R. ....Ayden
Edwards, Mrs. Vertie D. ....Stokesdale
Egerton, Mrs. Courtney David
2528 York Rd. ....Raleigh
Elesha, Mrs. William
3040 Briarclffe Rd. ....Winston-Salem

Elfmon, Mrs. Samuel Leon
117 Stedman St. .... Fayetteville
Ellington, Mrs. Amzi Jefferson, Jr.
419 Fountain Place .... Burlington
Elliott, Mrs. Avon Hall
607 Colonial Drive .... Wilmington
Elliott, Mrs. John Palmer
Greenway Drive .... Spray
Elliott, Mrs. Joseph Alexander, Sr.
2700 Sherwood Ave .... Charlotte
Elliott, Mrs. Joseph Alexander, Jr.
2224 Sanford Lane .... Charlotte
Epple, Mrs. Kenneth H.
1518 Liberty Drive .... Greensboro
Erb, Mrs. Norris Scribner
8 Oak Road .... Salisbury
Erdman, Mrs. Lawrence Huntington
P. O. Box 283 .... Bridgeton
Ernst, Mrs. Henry Edwin
97 Ingleside Dr. .... Concord
Ervin, Mrs. John Witherspoon
518 Lenoir St. .... Morganton
Erwin, Mrs. Evan Alexander, Jr.
516 S. Main .... Laurinburg
Espey, Mrs. Dan, Jr.
454 5th St., S.E. .... Hickory
Estes, Mrs. Edward Harvey, Jr.
3542 Hamstead Court .... Durham
Etherington, Mrs. John Lawrence
1703 Evergreen Ave. .... Goldsboro
Eubanks, Mrs. William Malcolm, Jr.
1700 Sterling Rd. .... Charlotte

Fagan, Mrs. Harry, Jr.
2508 Oxford Rd. .... Raleigh
Fail, Mrs. Phillip J.
Chestnut Drive .... Blowing Rock
Faison, Mrs. Elias Sampson
1825 Providence Rd. .... Charlotte
Fales, Mrs. Robert Martin
153 Renovah Circle .... Wilmington
Falls, Mrs. Fred
855 W. Marion St. .... Shelby
Falvo, Mrs. Samuel Catanzaro
716 Maybank Drive .... Hendersonville
Farley, Mrs. William Winfree
2625 Dover Rd. .... Raleigh
Farmer, Mrs. Thomas Wohlsen
1304 Mason Farm Rd. .... Chapel Hill
Farmer, Mrs. William Anderson
2841 Skye Dr. .... Fayetteville
Farmer, Mrs. William Dempsey
1011 Country Club Dr. .... Greensboro
Farmer, Mrs. Woodard Eason
27 Park Road .... Asheville
Faulk, Mrs. James Grady
1208 E. Franklin .... Monroe
Feezor, Mrs. Charles Noel
6 Pine Tree Rd. .... Salisbury
Feldman, Mrs. Leon Henry
6 N. Kensington Rd. .... Asheville
Felton, Mrs. Robert Lee, Jr.
Box 176 .... Carthage
Felts, Mrs. John Harvey, Jr.
3335 Paddington Lane .... Winston-Salem
Fender, Mrs. James Earle .... Waynesville
Ferguson, Mrs. George Burton
3938 Dover Rd., Hope Valley .... Durham
Ferrell, Mrs. John Atkinson
Apt. 8-B, Carolina Hotel .... Raleigh
Fesperman, Mrs. Joseph Claude
Box 517 .... Stanley
Fetter, Mrs. Bernard Frank
Summerset Drive .... Durham
Feuer, Mrs. Abe Lawrence
1006 Fairfield Dr. .... Gastonia
Fewell, Mrs. Richard Alexander
506 Hillcrest Rd. .... Burlington
Ficklin, Mrs. Conway
730 Princeton Dr. .... Wilmington
Field, Mrs. Bob Lewis
Box 557 .... Salisbury
Fields, Mrs. Leonard Earl
Box 788, Hidden Hills .... Chapel Hill
Fike, Mrs. Ralph Llewellyn
901 Raleigh Rd. .... Wilson
Finley, Mrs. Charles Francis
2323 Morganton Rd. .... Fayetteville
Fish, Mrs. Harry Gustav, Jr.
1116 Long Ave. .... Rocky Mount
Fisher, Mrs. George Walton, Jr.
2612 Edgewater Dr. .... Fayetteville
Fiske, Mrs. Reginald Earle
250 McCoy Road .... Salisbury
Fitz, Mrs. Thomas Edmunds
423 10th St. Dr. .... Hickory
Fitzgerald, Mrs. Charles Edmund
415 E. Wilson St. .... Farmville
Fitzgerald, Mrs. John Dean
210 Crestwood Dr. .... Roxboro
Fitzgerald, Mrs. John Hill, Jr.
217 Buff St. .... Lincolnton
Fitzgerald, Mrs. Robert Greeson, Jr.
320 S. Lamar St. .... Roxboro
Fleetwood, Mrs. Joseph Anderton, Sr. .... Conway
Fleetwood, Mrs. Joseph Anderton, Jr. .... Conway
Fleishman, Mrs. Malcolm
130 Herndon St. .... Fayetteville
Fleming, Mrs. Lawrence Edwin
1116 Providence Rd. .... Charlotte
Fleming, Mrs. Major Ivy
104 S. Franklin St. .... Rocky Mount
Fleming, Mrs. Paul A.
440 Peyton St. .... Raleigh
Fleming, Mrs. Ralph Gibson
23 Beverly Dr. .... Durham
Fleming, Mrs. William LeRoy .... Chapel Hill
Flowe, Mrs. Benjamin Hugh
33 Summit Ave. .... Concord
Floyd, Mrs. Anderson Gayle
N. Thompson St. .... Whiteville
Floyd, Mrs. Hal Stanfield
Lake View Rd. .... Fairmont
Floyd, Mrs. Walter
2011 Woodrow St. .... Durham

Floyd, Mrs. William Russel
Mt. Pleasant Highway .... Concord

Flythe, Mrs. William Henry
809 Hillcrest Dr. .... High Point

Foard, Mrs. Fred T., Jr.
1815 W. Smallwood Dr. .... Raleigh

Fogleman, Mrs. Ross Lee, Jr.
904 W. Highland Ave. .... Kinston

Follo, Mrs. Paige Bill
1709 Efland Dr. .... Greensboro

Fondren, Mrs. Frank
302 Jackson St. .... Roanoke Rapids

Forbes, Mrs. Gus Evans
Park Circle .... Laurinburg

Forbes, Mrs. Thomas Earl
Country Club Drive .... Reidsville

Ford, Mrs. David Emerson
103 Bridge St. .... Washington

Forrest, Mrs. William W.
1001 Montpelier Dr. .... Greensboro

Forsyth, Mrs. Harry Francis, Jr.
434 Westview Dr. .... Winston-Salem

Fortescue, Mrs. William Nicholas
Kanuga Rd. .... Hendersonville

Fortney, Mrs. Austin Powell

Fortune, Mrs. Benjamin Fletcher .... Jamestown
906 Cornwallis Dr. .... Greensboro

Foster, Mrs. Bobby .... Mocksville

Foster, Mrs. Clarence B.
East Indiana Ave. Ext. .... Southern Pines

Foster, Mrs. John W.
294 W. End Blvd. .... Winston-Salem

Foster, Mrs. Malcolm Tennyson
114 Stedman St. .... Fayetteville

Foushee, Mrs. J. Henry Smith, Jr.
748 Barnesdale Rd. .... Winston-Salem

Fowler, Mrs. Henry Jackson
Box 416 .... Walnut Cove

Fowler, Mrs. John A.
1212 Woodburn Rd. .... Durham

Fox, Mrs. Dennis Bryan
McGill Dr. .... Albemarle

Fox, Mrs. Norman Albright, Sr.
Friendly Rd. .... Guilford College

Fox, Mrs. Norman Albright, Jr.
Friendly Rd. .... Guilford College

Fox, Mrs. Powell Graham
2910 Fairview Rd. .... Raleigh

Fox, Mrs. Powell Graham, Jr.
3013 Medlin Drive .... Raleigh

Fox, Mrs. Robert Eugene
1011 E. Main St. .... Albemarle

Fox, Mrs. William Morgan
435 Charlotte Dr. .... Fayetteville

Fraasa, Mrs. Robert Conrad
1226 Tarrington Dr. .... Charlotte

Frankl, Mrs. George
923 Arbor Road .... Winston-Salem

Franklin, Mrs. Ernest Washington
1141 Linganore Pl. .... Charlotte

Franz, Mrs. Bruce Johnston
51 Sunset Parkway .... Asheville

Frazer, Mrs. Joe W., Jr.
2210 Hathaway Dr. .... Greensboro

Frazier, Mrs. Claude Albee
14 Buena Vista Rd. .... Asheville

Frazier, Mrs. John Wesley, Jr.
Pine Tree Rd. .... Salisbury

Freeman, Mrs. Percy Lee
1018 Paramount Circle .... Gastonia

Freeman, Mrs. Roy Oscar .... Jefferson

Freeman, Mrs. William Harrison
611 Yadkin St. .... Albemarle

Freeman, Mrs. William Talmadge
311 Vanderbilt Rd. .... Asheville

Fritz, Mrs. Olin Grady .... Walkertown

Fritz, Mrs. William Abel
636 Third St., N. E. .... Hickory

Frizelle, Mrs. Mark Twain
507 S. Lee St. .... Ayden

Frye, Mrs. Glenn Raymer
539 N. Center St. .... Hickory

Fulcher, Mrs. Luther .... Beaufort

Fuller, Mrs. Henry Fleming
1302 Walker Dr. .... Kinston

Fulp, Mrs. James Francis
Bryan Street .... Stoneville

Furgurson, Mrs. Ernest Whitmal .... Plymouth

Gadd, Mrs. Duwayne Douglas
Green Dial Cottage .... Pinehurst

Gainey, Mrs. John White, Jr.
Country Club Rd. .... Morehead City

Gallant, Mrs. Robert Miller
809 Central Ave. .... Charlotte

Galloway, Mrs. George .... Hamlet

Galloway, Mrs. James Hervey
200 Shepherd St. .... Raleigh

Galusha, Mrs. Bryant Leroy
6053 Brookhaven Rd. .... Charlotte

Gamble, Mrs. John Reeves, Jr.
Box 270 .... Lincolnton

Gambrel, Mrs. Ralph
546 Wilkesboro St. .... Mocksville

Garber, Mrs. Edgar Clyde, Jr.
1810 Lakeshore Dr. .... Fayetteville

Gardner, Mrs. F. Sidney
606 Forest Lake Rd. .... Fayetteville

Garrard, Mrs. Robert Lemley
101 N. Park Dr. .... Greensboro

Garrenton, Mrs. Connell George .... Bethel

Garrett, Mrs. John Bostian .... Walkertown

Garrett, Mrs. Norman Hessen, Jr.
3932 Madison Ave. .... Greensboro

Garrison, Mrs. Paul Leslie
1837 Buena Vista .... Winston-Salem

Garrison, Mrs. Ralph Bernard
Cheraw Rd. .... Hamlet

Garrison, Mrs. Robert Lee
2118 Beverly Dr. .... Charlotte

Garvey, Mrs. Fred Kesler
440 Fairfax Dr. .... Winston-Salem

Garvey, Mrs. Robert Roby
Boone Highway .... Blowing Rock

Gaskin, Mrs. Ernest Reed
100 Nottingham Dr. ....Charlotte

Gaskin, Mrs. Lewis Reid
274 N. Fourth St. ....Albemarle

Gaskin, Dr. Madge Baker
265 N. Third St. ....Albemarle

Gaul, Mrs. John Stuart, Sr.
2119 Norton Rd. ....Charlotte

Gaul, Mrs. John Stuart, Jr.
2010 Sharon Lane ....Charlotte

Gaul, Mrs. Rufus Wharton
3012 Hampton Ave. ....Charlotte

Gay, Mrs. Charles H.
143 Huntley Pl. ....Charlotte

Geddie, Mrs. Kenneth Baxter
1121 Rotary Dr. ....High Point

Gee, Mrs. William N., Jr.
1001 S. Madison ....Goldsboro

Gentry, Mrs. George Wesley
607 S. Main St. ....Roxboro

Gentry, Mrs. George Wesley, Jr.
Club Lake Street ....Roxboro

Gentry, Mrs. William Harold
N. C. Sanatorium ....McCain

Georgiade, Mrs. Nicholas G.
2523 Wrightwood Ave. ....Durham

Gerkens, Mrs. Gerke J. A. d'Armaud
N. C. Sanatorium ....McCain

Germouth, Mrs. Frederick George, Jr.
2100 Sherwood Ave. ....Charlotte

Gibbon, Mrs. James Wilson
720 Bromley Rd. ....Charlotte

Gibbons, Mrs. Julius Joyce, Jr.
215 Highland Ave. ....Lenoir

Gibbs, Mrs. Norfleet M.
209 Johnson St. ....New Bern

Gibbs, Mrs. Stuart Wynn
Armstrong Park Circle ....Gastonia

Gibson, Mrs. Milton Reynolds
2533 York Rd. ....Raleigh

Gibson, Mrs. Thomas G., Jr.
Main St. ....Gibson

Gill, Mrs. Joseph Armstrong
803 River Rd. ....Elizabeth City

Gilliam Mrs. Charles F.
15 Fifth Ave. ....Thomasville

Gilmore, Mrs. Clyde Manly
108 E. Avondale ....Greensboro

Gilmour, Mrs. Monroe Taylor
734 Granville Rd. ....Charlotte

Givens, Mrs. George H., Jr. ....Taylorsville

Glasgow, Mrs. Douglas McKay
2022 Glendale Rd. ....Charlotte

Glasson, Mrs. John
615 Swift Ave. ....Durham

Glenn, Mrs. Channing
Box 335 ....Elizabethtown

Glenn, Mrs. Henry Franklin, Jr.
319 S. Oakland Ave. ....Gastonia

Glenn, Mrs. John C., Jr.
200 Hempstead Pl. ....Charlotte

Glenn, Mrs. Richard Reece
2507 Miller Park Cl. ....Winston-Salem

Gobble, Mrs. Flettus Lee, Jr.
925 S. Hawthorne Rd. ....Winston-Salem

Gobel, Mrs. William Kenneth ....Denton

Gold, Mrs. Ben Miller, Jr.
Country Club Dr. ....Rocky Mount

Goldner, Mrs. J. Leonard
602 E. Forest Hills Blvd. ....Durham

Goley, Mrs. Willard Coe
217 N. Main St. ....Graham

Goodman, Mrs. Benjamin Warren
226 Fifth St., S. E. ....Hickory

Goodwin, Mrs. Cleon Walton
1107 W. Nash St. ....Wilson

Goodwin, Mrs. Oscar Sexton
Raleigh Rd. ....Apex

Googe, Mrs. James Turner
335 Grand Boulevard ....Boone

Gore, Mrs. John Pratt
3208 Denise St. ....Durham

Goree, Mrs. John A.
3817 Hillgrand Cr. ....Durham

Goswick, Mrs. Claude Benjamin
1747 Clairmont Drive ....Hendersonville

Goswick, Mrs. Harry Wilson, Jr.
280 Canterbury Trail ....Winston-Salem

Gradis, Mrs. Howard Henry
Forest Hill Drive ....Greenville

Grady, Mrs. Franklin McLean
Madam Moore's Lane ....New Bern

Graham, Mrs. Charles Pattison
123 Forest Hills Dr. ....Wilmington

Graham, Mrs. David Eric
3626 Stonehaven Dr. ....Charlotte

Graham, Mrs. Walter Raleigh
741 Hempstead Pl. ....Charlotte

Graham, Mrs. William Alexander
2247 Cranford Rd. ....Durham

Gray, Mrs. Cyrus Leighton
912 Rotary Dr. ....High Point

Green, Mrs. Harold David
1172 Hawthorne Rd. ....Winston-Salem

Green, Mrs. Paul, Jr.
1353 Parkview Cr. ....Salisbury

Green, Mrs. Philip Palmer
435 E. Indiana Ave. ....Southern Pines

Greene, Mrs. Phares Yates
1004 E. Willowbrook Dr. ....Burlington

Greene, Mrs. William Alexander
500 Pinkney St. ....Whiteville

Greenwood, Mrs. James Brooks, Jr.
2319 Providence Rd. ....Charlotte

Gregg, Mrs. Robert A.
3411 Cloverdale Dr. ....Greensboro

Gregory, Mrs. John Eugene
521 Confederate Ave. ....Salisbury

Gregory, Mrs. R. D., Jr.
105 Kimberly Knoll ....Asheville

Gregory, Mrs. William Lyon
120 Easton Burton Hill ....Lowell

Greiss, Mrs. Frank
1041 Watson Ave. ....Winston-Salem

Gridley, Mrs. Timothy H.
820 Carolina Ave. ....Fayetteville

Grier, Mrs. Charles Talmadge
Box 475 .... Carthage
Grier, Mrs. John Calvin, Jr.
Midland Road .... Pinehurst
Griffin, Mrs. Harold Walker
537 N. Center St. .... Hickory
Griffin, Mrs. Robert Ashley
11 Hilltop Rd. .... Asheville
Griffin, Mrs. Thomas Ray,
Box 328 .... Troutman
Griffin, Mrs. William Ray, Jr.
30 Hilltop Rd. .... Asheville
Griffis, Mrs. John Williams
Box 191 .... Denton
Griggs, Mrs. Willard Wilson
Box 217 .... Norwood
Grim, Mrs. Kenneth Boyd
1421 Broad St. .... Durham
Grimmett, Mrs. Matthew Hill
107 Country Club Dr. .... Concord
Groat, Mrs. Richard A.
3603 Kirby Drive .... Greensboro
Groome, Mrs. James Gordon
203 Edgedale Dr. .... High Point
Gross, Mrs. Francis Warren
408 W. Lexington Ave. .... High Point
Grove, Mrs. Raymond Fisk
1400 Live Oak Pkwy. .... Wilmington
Groves, Mrs. Robert Burwell, Sr. .... Lowell
Groves, Mrs. Robert Burwell, Jr.
2565 Pinewood Drive .... Gastonia
Gulley, Mrs. Marcus Marcellus
1836 Runnymeade Rd. .... Winston-Salem
Gunn, Mrs. Charles Groshon
972 Kenleigh Circle .... Winston-Salem
Gunter, Mrs. Arthur Rhett
2127 Radcliffe Ave. .... Charlotte
Gunter, Mrs. June U.
1411 N. Mangum St. .... Durham
Gwynn, Mrs. Houston Lafayette
Box 6 ________ Yanceyville

Haar, Mrs. Frederick Behrend
608 E. 9th St. .... Greenville
Hackney, Mrs. Ben H. .... Lucama
Hadley, Mrs. Herbert Wood
2607 S. Dickinson Ave. .... Greenville
Hagaman, Mrs. John Bartlett, Jr.
304 North St. .... Boone
Hagaman, Mrs. Len Doughton
101 Cherry Dr. .... Boone
Haines, Mrs. Hilton Drummond
700 E. Washington St. .... Rockingham
Haines, Mrs. Innes Correll
Rt. 4, Box 563 .... Fayetteville
Hairfield, Mrs. Beverly Dew
415 W. Union St. .... Morganton
Hairfield, Mrs. Theodore Vincent
404 Westview St. .... Lenoir
Hall, Mrs. James Brownlee
Rt. 1, Box 348-A ________ Matthews
Hall, Mrs. John Moir
Box 763 ________ Elkin
Hall, Mrs. Joseph Cullen
839 Fairmont Ave. .... Salisbury
Hall, Mrs. William Bruce, Jr.
543 Vista Dr. .... Fayetteville
Hall, Mrs. William Dewey
510 Washington St. .... Roanoke Rapids
Hall, Mrs. William Hugh
3635 Barclay Downs Dr. .... Charlotte
Ham, Mrs. Clem
709 West Blvd. .... Laurinburg
Ham, Mrs. George Calverno
519 Dogwood Dr. .... Chapel Hill
Hambrick, Mrs. Robert Theodore
529 Sixth St., N. W. .... Hickory
Hambright, Mrs. Rufus Roberts
2322 Danbury Rd. .... Greensboro
Hamer, Mrs. Alfred Wilson
112 Pearson Dr. .... Morganton
Hamer, Mrs. Douglas, Jr.
205 Norwood St. .... Lenoir
Hamer, Mrs. Eugene Floyd
P. O. Box 476 .... Monroe
Hamilton, Mrs. Alfred Thomason
1422 Canterbury Rd. .... Raleigh
Hamilton, Mrs. Frank Hutchinson, Jr.
2815 Marlowe Dr. .... Charlotte
Hamilton, Mrs. John Homer
2124 Cowper Dr. .... Raleigh
Hamilton, Mrs. Joseph Franklin, Jr.
Albemarle Park ________ Asheville
Hammond, Mrs. Alfred Franklin, Jr.
1514 Neuse Blvd. .... New Bern
Hampton, Mrs. James H. .... Lewisville
Hamrick, Mrs. John Carl
1002 Kings Rd. .... Shelby
Hamrick, Mrs. Ladd Watts, Jr.
Northeast Drive .... Concord
Hand, Mrs. Edgar Hall .... Pineville
Hand, Mrs. LeRoy Corbett, Jr. .... Gatesville
Hanes, Mrs. Gideon Isaac, Jr.
836 Wellington Road .... Winston-Salem
Happer, Mrs. William
205 Woodsway Lane .... Lenoir
Harbison, Mrs. John William
911 N. Washington St. .... Shelby
Hardaway, Mrs. John Stegar
434 Oakwood Dr. .... Statesville
Harden, Mrs. Boyd
Alamance Road .... Burlington
Hardin, Mrs. Eugene Ramsey
1103 N. Elm St. .... Lumberton
Hardin, Mrs. Richard Henry
205 S. Granville St. .... Edenton
Hardin, Mrs. Ronda Horton
Wilkesboro Rd. .... Boone
Hardison, Mrs. Lewis Benjamin
113 Star Hill Rd. .... Fayetteville
Hardman, Mrs. Edward Francis
Route 2, Huntington Park .... Charlotte
Hare, Mrs. R. Bryant, Jr.
Masonboro Sound .... Wilmington
Hare, Mrs. Roy Allen
1023 Sycamore St. .... Durham

Harer, Mrs. Adolph Eugene
1609 Canterbury Rd. .... Raleigh
Harloe, Mrs. John Pinckney
669 Hempstead Pl. .... Charlotte
Harmon, Mrs. Raymond Harris
Highland Dr. .... Boone
Harper, Mrs. Matt C., Jr.
Caswell Training School .... Kinston
Harper, Mrs. Robert N.
3322 Ocatea Drive .... Raleigh
Harrell, Mrs. William Fletcher, Jr.
Brother's Dr. .... Elizabeth City
Harrill, Mrs. Henry Clay
100 Elmwood Terrace .... Greensboro
Harrill, Mrs. James Albert
2860 Reynolds Rd. .... Winston-Salem
Harrington, Mrs. Lee I., Jr.
2423 Fairway Dr. .... Winston-Salem
Harris, Mrs. Julian L.
1660 Mansfield Rd. .... Winston-Salem
Harris, Mrs. Loftin Howell
417 East St. .... Albemarle
Harry, Mrs. John McKamie
832 W. Rowan St. .... Fayetteville
Hart, Mrs. Julian Deryl
2324 Duke University Rd. .... Durham
Hart, Mrs. Lillard Franklin
236 E. Olive .... Apex
Hart, Mrs. Oliver James
1930 Georgia Ave. .... Winston-Salem
Hart, Mrs. Verling Kersey
106 W. 7th St. .... Charlotte
Hartman, Mrs. Bernhard Henry
12 Cambridge Rd. .... Asheville
Hartness, Mrs. William Rufus, Jr.
615 Carr St. .... Sanford
Harvey, Mrs. Wallace Watson, Jr. .... Manteo
Hatcher, Mrs. Samuel W. .... Morehead City
Hawes, Mrs. Cecil Jennings
2101 Wendover Rd. .... Charlotte
Hawes, Mrs. George Aubrey
1862 Queens Rd. W. .... Charlotte
Hawes, Mrs. James Beebe
Mimosa Shores .... Washington
Hawkins, Mrs. Barry Fugh
Williamsburg Road .... Concord
Hawkins, Mrs. Hal Burgess .... Moravian Falls
Hawkins, Mrs. James Hubert .... Alamance
Hayes, Mrs. Donald Michael
1324 Irving St. .... Winston-Salem
Hayes, Mrs. Hugh Harrison, Jr.
3212 Country Club Dr. .... Charlotte
Hayes, Mrs. James Willard
Lake View Rd. .... Fairmont
Hayes, Mrs. William Clayton
Woodland Blvd. .... Wilkesboro
Haywood, Mrs. Hubert Benbury, Jr.
2718 Gloucester Rd. .... Raleigh
Heafner, Mrs. Bob O.
Taylorsville Road .... Stony Point
Hedgepeth, Mrs. Emmett Martin
Crestwood Dr. .... Roxboro
Hedgpeth, Mrs. Edward McGowan
Rt. 3, Box 87 .... Chapel Hill
Hedgpeth, Mrs. Louten Rhodes
1917 N. Walnut St. .... Lumberton
Hedgpeth, Mrs. William Carey
2405 Kenan St. .... Lumberton
Hedrick, Mrs. Clyde Reitzel
318 E. College Ave. .... Lenoir
Hedrick, Mrs. Richard Eli
1999 Georgia Ave. .... Winston-Salem
Hege, Mrs. John Roy
905 Martin Drive .... Concord
Heinitsh, Mrs. George W.
Knollwood .... Southern Pines
Helms, Mrs. Jefferson Bivins
319 W. Union St. .... Morganton
Helsabeck, Mrs. Belmont Augustus
2315 Country Club Rd. .... Winston-Salem
Helsabeck, Mrs. Chester Joseph
Box 403 .... Walnut Cove
Helsabeck, Mrs. Rupert Sylvester
2080 Polo Road, N. W. .... Winston-Salem
Hemmings, Mrs. Hugh Carroll
Lurawood Dr. .... Morganton
Hemphill, Mrs. James Eugene
2002 Pinewood Circle .... Charlotte
Henderson, Mrs. John Percy, Sr.
417 College St. .... Jacksonville
Hendricks, Mrs. Paul Eugene
808 W. Mountain St. .... Kings Mountain
Henry, Mrs. Hector H.
3535 Providence Rd. .... Charlotte
Henschen, Mrs. Hal
1309 General Lee Ave. .... Fayetteville
Henson, Mrs. Thomas Albert
1105 Country Club Dr. .... Greensboro
Herrin, Mrs. Keith Hermon
1204 Fairfield Dr. .... Gastonia
Herring, Mrs. Theodore Tilghman
Ripley Road .... Wilson
Herring, Mrs. William Benjamin
Carolyn Drive .... Albemarle
Hester, Mrs. Joseph Robert
1 Buffalo St. .... Wendell
Hester, Mrs. William Shepherd
802 Main St. .... Reidsville
Hewitt, Mrs. Willard Chappel
W. Front St. Ext. .... Burlington
Hiatt, Mrs. Joseph Spurgeon, Jr.
Box 85 .... Southern Pines
Hickman, Mrs. Harry Stuart
Hickory Highway .... Lenoir
Hicks, Mrs. Vonnie Monroe, Jr.
1515 Scales St. .... Raleigh
Higgins, Mrs. Robert Donald
Box 6065, Five Points Sta. .... Raleigh
High, Mrs. Larry Allson .... Nashville
Highsmith, Mrs. Charles, Sr.
210 N. Wilson Ave. .... Dunn
Highsmith, Mrs. Charles, Jr. .... Troy
Highsmith, Mrs. George Perry
Valley Road .... Thomasville

Highsmith, Mrs. William Cochran
220 Bradford Ave. ....Fayetteville
Highsmith, Mrs. William Jesse, Jr.
Box 671 ....Plymouth
Hightower, Mrs. Felda
2455 Reynolds Drive ....Winston-Salem
Hilderman, Mrs. Walter Carrington, Jr.
1724 Brandon Rd. ....Charlotte
Hill, Mrs. Millard D.
818 Daniels St. ....Raleigh
Hill, Mrs. William Henry
115 E. South St. ....Albemarle
Hinman, Mrs. Alanson
792 Roslyn Rd. ....Winston-Salem
Hipp, Mrs. Edward Reginald, Sr.
348 Hempstead Pl. ....Charlotte
Hitch, Mrs. Joseph Martin
918 Cowper Dr. ....Raleigh
Hobart, Mrs. Seth Guilford, Jr.
2011 W. Club Blvd. ....Durham
Hodges, Mrs. Horace Hayden
423 Ferncliff Rd. ....Charlotte
Hoggard, Mrs. William Alden, Jr.
2501 Rochelle ....Elizabeth City
Hogshead, Mrs. Ralph, Jr.
230 W. Park Dr. ....Morganton
Hoke, Mrs. Harold Reid
1605 Oaklawn Dr. ....Greenville
Holbrook, Mrs. Joseph Samuel
223 N. Oak St. ....Statesville
Holbrook, Mrs. William Douglas
4141 Arborway ....Charlotte
Hollandsworth, Mrs. Luther Clarence
305 E. 18th St. ....Lumberton
Hollister, Mrs. William Fredwin
Midland Rd. ....Southern Pines
Hollowell, Mrs. Victor Boyce
515 Fenton Pl. ....Charlotte
Hollyday, Mrs. William Murray
51 Lawrence Pl. ....Asheville
Holmes, Mrs. George Washington
524 Roslyn Rd. ....Winston-Salem
Holt, Mrs. Lawrence Byerly
2812 Reynolds Dr. ....Winston-Salem
Hood, Mrs. Christopher Kennedy
5143 Beckford Dr. ....Charlotte
Hood, Mrs. Richard Thornton, Jr.
1502 Rosedale ....Kinston
Hooks, Mrs. Richard Eugene ....St. Pauls
Hooper, Mrs. Clifford Harold
20 Lynnstone Court ....Asheville
Hooper, Mrs. Joseph Ward, Sr.
1817 Market St. ....Wilmington
Hooper, Mrs. Joseph Ward, Jr.
2600 Parmelee Dr. ....Wilmington
Hoot, Mrs. Melvin Phillip
1505 E. 5th St. ....Greenville
Horner, Mrs. Jack Chenoweth ....Spruce Pine
Hornowski, Mrs. Marcel Jerome
317 Charlotte St. ....Asheville
Horsley, Mrs. William Nolen
South Point Rd. ....Belmont
Horsley, Mrs. Thomas Martin ....Elizabeth City
Hoskins, Mrs. John Robinson, III
36 Evelyn Pl. ....Asheville
Hoskins, Mrs. William Hume
Fuller St. ....Whiteville
Hough, Mrs. Mac Johnson
3234 Park Rd. ....Charlotte
Houghton, Mrs. Raymond C.
1800 River Dr. ....New Bern
Houser, Mrs. Forest Melville
Elm St. ....Cherryville
Howard, Mrs. Corbett Etheridge
618 E. Park Ave. ....Goldsboro
Howard, Mrs. Joseph Cooper, Jr.
407 Lafayette St. ....Clinton
Howard, Mrs. Paul Osman
Carbonton Hgts. ....Sanford
Howell, Mrs. Charles Maitland, Jr.
1100 Kent Road, East ....Winston-Salem
Howell, Mrs. Julius Ammons
2662 Robin Hood Rd. ....Winston-Salem
Howell, Mrs. William Lawrence ....Ellerbe
Hubbard, Mrs. Frederick Cecil, Sr. ....Wilkesboro
Hubbard, Mrs. Robert Thomas
126 Lakeshore Dr. ....Asheville
Huckeriede, Mrs. Mark Henry
McNeill Drive ....Laurinburg
Hudson, Mrs. Miles Hildebrand
240 Bouchard St. ....Valdese
Huey, Mrs. Thomas Walker, Jr.
2438 Sharon Rd. ....Charlotte
Huffman, Mrs. Stanton Vance
Route 2 ....Elon College
Hughes, Mrs. Carlisle Bee, Jr.
Box 326 ....Yadkinville
Hughes, Mrs. Jack
1004 Demerius ....Durham
Humphries, Mrs. Charles Oliver
3835 Sommerset Road ....Durham
Huneycutt, Mrs. Joel Broadus
627 Yadkin St. ....Albemarle
Hunt, Mrs. Jasper Stewart
2064 Queens Rd., E. ....Charlotte
Hunt, Mrs. Walter Skellie, Jr.
1606 Canterbury Rd. ....Raleigh
Hunt, Mrs. William Jack
720 Ferndale Dr. ....High Point
Hunter, Mrs. John B.
618 E. Marion St. ....Shelby
Hunter, Mrs. John Pullen
325 S. Academy St. ....Cary
Hunter, Mrs. W. Myers
800 E. Blvd. ....Charlotte
Hunter, Mrs. William Blair
1007 10th St. ....Lillington
Hunter, Mrs. William Cooper
1106 W. Nash St. ....Wilson
Hurdle, Mrs. Samuel Walker
2571 Country Club Rd. ....Winston-Salem
Hurdle, Mrs. Thomas Gray
2503 Mirror Lake Dr. ....Fayetteville
Hutchinson, Mrs. Sankey Smith ....Bladenboro

Ingram, Mrs. Charles Hal
1105 Rotary Dr. .... High Point
Inman, Mrs. Charles Ernest
Fisher Park .... Fairmont
Irving, Mrs. Richard Carroll
601 4th Ave. W. .... Hendersonville
Irwin, Mrs. Henderson .... Eureka
Isenhower, Mrs. Joseph Andrew
232 Fifth St., S. E. .... Hickory
Ivey, Mrs. Henry B.
105 N. Pineview Ave. .... Goldsboro
Jackson, Mrs. Richard DeWitt
Box 548 .... Mt. Airy
Jackson, Mrs. Robert Toombus
3347 Alamance Dr. .... Raleigh
Jackson, Mrs. Roger A.
111-A Dobbin Ave. .... Fayetteville
Jacobs, Mrs. Julian Erich John
2000 Providence Rd. .... Charlotte
James, Mrs. Arthur Augustus, Jr.
614 Spring Lane .... Sanford
James, Mrs. George W.
1020 Wellington Rd. .... Winston-Salem
James, Mrs. Richard Thomas, Jr.
4051 Abingdon Rd. .... Charlotte
James, Mrs. William Daniel
Vance St. .... Hamlet
James, Mrs. William Duer, Jr.
306 Entwistle St. .... Hamlet
Jarman, Mrs. Fontaine Graham, Sr.
402 Hamilton St. .... Roanoke Rapids
Jarman, Mrs. Fontaine Graham, Jr.
Rochelle Court .... Roanoke Rapids
Jarrell, Mrs. Wilburn Eric
329 Country Club Rd. .... Mt. Airy
Jarvis, Mrs. James Luther
1003 Woodland Drive .... Gastonia
Jenkins, Mrs. Albert Milton
823 Bryan St. .... Raleigh
Jennings, Mrs. Royal Green
724 Florham Ave. .... High Point
Jervey, Mrs. William St. Julien
907 Elizabeth Rd. .... Shelby
Jeter, Mrs. Robert Vernon .... Plymouth
Johnson, Mrs. Amos Neill .... Garland
Johnson, Mrs. Cecil L.
1616 Palm St. .... Goldsboro
Johnson, Mrs. Charles Thomas, Jr. .... Red Springs
Johnson, Mrs. Charles Thomas, Sr. .... Red Springs
Johnson, Mrs. Floyd
201 Pinkney St. .... Whiteville
Johnson, Mrs. Gale Denning
400 W. Broad St. .... Dunn
Johnson, Mrs. Gaston Frank
3225 Nottingham Rd. .... Winston-Salem
Johnson, Mrs. George, Jr.
1312 Watts St. .... Durham
Johnson, Mrs. Harry Lester
Box 530 .... Elkin
Johnson, Mrs. Harry L., Jr. .... Granite Quarry
Johnson, Mrs. Heber Wellington
3002 Wayne Dr. .... Wilmington
Johnson, Mrs. James Trimble
312 E. 16th .... Lumberton
Johnson, Mrs. John Ralph
N. Orange St. .... Dunn
Johnson, Mrs. Joseph A.
Winslow Acres .... Elizabeth City
Johnson, Mrs. Joseph Lewis
205 N. Main St. .... Graham
Johnson, Mrs. Paul William
Route 8, Green Meadows .... Winston-Salem
Johnson, Mrs. Robert Charles
701 Locust St. .... High Point
Johnson, Mrs. Walter Royle
3 Fairway Place .... Asheville
Johnson, Mrs. Wingate Memory
428 Stratford Rd. .... Winston-Salem
Johnston, Mrs. Frank Randolph
735 Arbor Rd. .... Winston-Salem
Johnston, Mrs. Harvey Wylie
1915 Club Rd. .... Charlotte
Johnston, Mrs. William Oliver
2611 Forest Dr. .... Charlotte
Jones, Mrs. A. McCray
Shady Banks .... Washington
Jones, Mrs. Beverly Nicholas, Sr.
455 Carolina Cl. .... Winston-Salem
Jones, Mrs. Beverly Nicholas, Jr.
633 Barnesdale Rd. .... Winston-Salem
Jones, Mrs. Clayton Joe
873 Arbor Lane .... Concord
Jones, Mrs. Craig Strickie
Westfield Road .... Shelby
Jones, Mrs. Dean Cicero .... Jefferson
Jones, Mrs. Edward L.
400 Randolph St. .... Thomasville
Jones, Mrs. Frank Woodson
Westlake Hills .... Newton
Jones, Mrs. Joseph Kempton
109 E. Boundary St. .... Chapel Hill
Jones, Mrs. Joseph Reid, Jr.
Box 298 .... King
Jones, Mrs. Martin Evans .... Granite Falls
Jones, Mrs. Otis Hunter
1710 Queens Rd. W. .... Charlotte
Jones, Mrs. Paul Erastus
Rt. 3, Box 452A .... Concord
Jones, Mrs. Robert Spurgeon
405 Beaumond .... Shelby
Jones, Mrs. Thomas Thweatt
2621 Stuart Dr. .... Durham
Jones, Mrs. William Isaac
3521 Highview Rd. .... Charlotte
Jones, Mrs. William Robert
217 Clifton Rd. .... Rocky Mount
Jordan, Mrs. John Alfred, Jr.
236 Pinecrest Dr. .... Fayetteville
Jordan, Mrs. Riley Moore
310 Fulton St. Ext. .... Raeford
Jordan, Mrs. Weldon Huske
601 Westmont Dr. .... Fayetteville
Joyce, Mrs. Charles Weldon
617 Decatur .... Madison

Joyner, Mrs. William Stafford
401 Whitehead Circle .... Chapel Hill
Judd, Mrs. Glenn Ballentine .... Varina
Justa, Mrs. Samuel Harry
505 Piedmont Ave. .... Rocky Mount
Justice, Mrs. William Shipp
14 White Oak Rd. .... Asheville
Justis, Mrs. Homer Rodeheaver
820 Fairbanks Rd. .... Charlotte

Kalevas, Mrs. Harry John
5415 Wedgewood Dr. .... Charlotte
Katz, Mrs. Joseph
Kinston Apts. .... Kinston
Kaufman, Mrs. Karl Frederick
S. Rugby Road .... Hendersonville
Kavanagh, Mrs. William Paul
1127 W. Henderson St. .... Salisbury
Kearns, Mrs. Paul Rutherford
640 Davie Ave. .... Statesville
Kearse, Mrs. William Oliver .... Canton
Keathley, Mrs. Franklin Burr
206 Grove Ave. .... Lenoir
Keever, Mrs. James Woodfin
623 Second Ave., N.W. .... Hickory
Keiter, Mrs. William Eugene
1507 Perry Park Dr. .... Kinston
Keith, Mrs. Julian Faison, Jr.
Box 635 .... Clarkton
Keith, Mrs. Marion Yates
1603 Carlisle Rd. .... Greensboro
Keleher, Mrs. Michael Francis
18 Maywood Rd. .... Asheville
Kelemen, Mrs. William Arthur
1206 Kennilworth Ave. .... Charlotte
Keller, Mrs. Guy Otis
1223 Providence .... Charlotte
Kelley, Mrs. Thomas Francis
805 Montgomery Ave. .... Albemarle
Kelly, Mrs. Luther Wrentmore, Sr.
1014 Kenilworth Ave. .... Charlotte
Kelly, Mrs. Luther Wrentmore, Jr.
3915 Suffolk Place .... Charlotte
Kelly, Mrs. Richard Alexander
308 N. Chapman .... Greensboro
Kemp, Mrs. Malcolm Drake
210 Highland Rd. .... Southern Pines
Kenan, Mrs. LeRoy Fulton
22 Henderson St. .... Badin
Kendall, Mrs. John Harold
800 Stewart Ave. .... Clinton
Kendrick, Mrs. Richard L.
2500 Roswell Ave. .... Charlotte
Kennard, Mrs. John William .... Hudson
Kennedy, Mrs. John Pressly
2026 Providence Rd. .... Charlotte
Kennedy, Mrs. Leon Toland
1907 Sterling Rd. .... Charlotte
Kent, Mrs. Alfred A., Jr. .... Granite Falls
Kermon, Mrs. Louis Todd
1625 Canterbury Rd. .... Raleigh
Kern, Mrs. John Campbell
Box 6 .... Booneville

Kernodle, Mrs. Charles Edward, Jr.
444 Tarleton Ave. .... Burlington
Kernodle, Mrs. Donald Reid
1521 Sherwood Dr. .... Burlington
Kernodle, Mrs. Dwight Talmadge
Route 2 .... Elon College
Kernodle, Mrs. George Wallace
518 Parkview Dr. .... Burlington
Kernodle, Mrs. Harold Barker
423 Glenwood Ave. .... Burlington
Kernodle, Mrs. John Robert
Edgewood Ave. Ext. .... Burlington
Kerns, Mrs. Thomas Cleveland, Sr.
120 Briar Cliff Rd. .... Durham
Kerns, Mrs. Thomas Cleveland, Jr.
1118 Wells St. .... Durham
Kerr, Mrs. George Russell
Woodland Ave. Ext. .... Burlington
Kerr, Mrs. John Guthrie .... Leicester
Kesler, Mrs. Robert Cicero
705 Twyckenham Dr. .... Greensboro
Kester, Mrs. John Marcas, Jr.
2935 Avondale Ave. .... Charlotte
Ketner, Mrs. Fred Yadkin
185 Washington Lane .... Concord
Keys, Mrs. Carson Meade .... West Jefferson
Kibler, Mrs. William Herbert
100 Valdese Ave. .... Morganton
Kidd, Mrs. Ralph Vincent, Jr.
227 Canterbury Rd. .... Charlotte
Killam, Mrs. A. Robert
3501 Sottywood Drive .... Fayetteville
Kimberly, Mrs. George D. .... Bakersville
Kimbrell, Mrs. Odell Culp
615 Currituck Dr. .... Raleigh
Kimel, Mrs. C. A.
4132 Snyder Dr. .... Winston-Salem
King, Mrs. Daniel
Belmont Dr. .... Reidsville
King, Mrs. Duncan I. Campbell .... Flat Rock
King, Mrs. Francis Parker
1603 Lucerne Way .... New Bern
King, Mrs. Parks McCombs
1419 Wendover Lane .... Charlotte
King, Mrs. Robert Rogers
138 Blowing Rock .... Boone
King, Mrs. Robert Wilson
113 Dobbin Ave. .... Fayetteville
King, Mrs. Walter Gorringe
1305 Latham Rd. .... Greensboro
Kinlaw, Mrs. Murray Carlyle
202 W. 21st St. .... Lumberton
Kirby, Mrs. William Leslie
734 Arbor Rd. .... Winston-Salem
Kirkland, Mrs. John Alvin
715 Trinity Dr. .... Wilson
Kirksey, Mrs. James Jackson
201 Riverside Dr. .... Morganton
Kirksey, Mrs. William Albert
302 S. King St. .... Morganton
Kistler, Mrs. Clark Clemmons
2212 St. Mary's St. .... Raleigh

Kitchen, Mrs. Thomas Ward
103 Meadowood Dr. ....Lenoir
Kitchin, Mrs. Thurman Delna
413 N. Main St. ....Wake Forest
Kitchin, Mrs. William Walton
Coharie Dr. ....Clinton
Klenner, Mrs. Fred Robert
1205 Forest Road ....Reidsville
Klostermyer, Mrs. Louis Leon
419 Vanderbilt Rd. ....Asheville
Kneedler, Mrs. William Harding
Box 397 ....Davidson
Knight, Mrs. Floyd Lafayette
Route 4 ....Sanford
Knoefel, Mrs. Arthur Eugene, Jr.
104 Laurel Circle ....Black Mountain
Knox, Mrs. Joseph Clyde
1228 S. Live Oak Parkway ....Wilmington
Kodack, Mrs. Albert
9 N. Kensington Rd. ....Asheville
Kokiko, Mrs. George Victor
110 Edgewood Rd. ....High Point
Koogler, Mrs. B. Robert ....Candor
Koonce, Mrs. Donald Brock
1407 Oleander Dr. ....Wilmington
Kornegay, Mrs. Lemuel Weyher, Jr. ....Warrenton
Koseruba, Mrs. George Michael
Rt. 1, Box 161-A ....Castle Hayne
Koury, Mrs. George Eli
Route 7, Cedarwood Drive ....Burlington
Kramer, Mrs. Morris
503 Walnut St. ....Lumberton
Kraycirik, Mrs. Emery Thomas
Alamance Acres ....Burlington
Kremers, Mrs. Marshall Y.
318 Westover Rd. ....Seymour Johnson Air Force Base
Kroh, Mrs. Laird Franklin
2201 McClintock Rd. ....Charlotte
Kroncke, Mrs. Fred George
623 Cedar St. ....Roanoke Rapids
Kurtz, Mrs. Elam ....Jefferson
Kutscher, Mrs. George William
29 Elk Mountain Scenic Hwy. ....Asheville
Kutteh, Mrs. Hanna Constantine
567 Lakeside Dr. ....Statesville
Kyles, Mrs. Norman Bruce
Cherry Hospital ....Goldsboro

Lackey, Mrs. Robert Stevenson
3931 Suffolk Pl. ....Charlotte
Lackey, Mrs. Walter Jackson ....Fallston
Lacy, Mrs. Thomas Allen
228 W. Fisher St. ....Salisbury
Lafferty, Mrs. John Ogden
1940 Overhill Rd. ....Charlotte
Lafferty, Mrs. John William
1055 Fourth Ave., N.W. ....Hickory
Lafferty, Mrs. Robert H.
1960 Randolph Rd. ....Charlotte
LaGrange, Mrs. C. Rex ....Bladenboro
Lahser, Mrs. Charles Irvin
Churchill Dr. ....Gastonia
Lake, Mrs. Ralph Callihan
106 Elgin Place ....Greensboro
Lambeth, Mrs. William Arnold, Jr.
Route 8 ....Winston-Salem
Lampley, Mrs. Charles Gordon
Fairway Dr. ....Shelby
Lampley, Mrs. William Askew
116 Briarwood Lane ....Hendersonville
Landon, Mrs. Henry C., III
611 Eighth St. ....North Wilkesboro
Lane, Mrs. Edgar Winslow, Jr.
Bouchard St. ....Valdese
Lang, Mrs. Andrew Martin
106 N. Anderson St. ....Morganton
Langdon, Mrs. Benjamin Bruce
Rt. 4, Box 567 ....Fayetteville
Lanier, Mrs. Verne Clifton
Box 75 ....Welcome
Lapsley, Mrs. Alberti Fraser
4 Tallassee St. ....Badin
Large, Mrs. Hiram Lee, Jr.
Route 1, Box 358-B ....Matthews
Larkin, Mrs. Ernest Wadill, Jr.
1202 Respass St. ....Washington
Lassiter, Mrs. Tallie E. ....Biscoe
Lassiter, Mrs. Will Hardee, Jr.
709 Sunset Dr. ....Smithfield
Latham, Mrs. Joseph Roscoe
1301 National Ave. ....New Bern
Laton, Mrs. James Franklin
116 E. North St. ....Albemarle
La Tourette, Mrs. Kenneth Abran ....Flat Rock
Lawing, Mrs. Karl Lander
327 N. Laurel St. ....Lincolnton
Lawrence, Mrs. Benjamin Jones, Jr.
S. 915 Rockford St. ....Mt. Airy
Lawrence, Mrs. John Charles
1200 N. Elm ....Lumberton
Lea, Mrs. James Walter, Jr.
721 W. Davis St. ....Burlington
Leath, Mrs. MacLean Bacon ....Archdale
Leath, Mrs. Thomas Edward
3632 Kempton Pl. ....Charlotte
LeBauer, Mrs. Maurice Leon
2023 St. Andrews Rd. ....Greensboro
Ledbetter, Mrs. John Winslow
919 Hendersonville Rd. ....Asheville
Lee, Mrs. Allen Henry
309 N. Massey ....Selma
Lee, Mrs. Ferdinand Wayne
442 Hempstead ....Charlotte
Lee, Mrs. Francis Brown
Pageland Rd. ....Monroe
Lee, Mrs. Thomas Leslie
Rountree St. ....Kinston
LeGrand, Mrs. Robert Hampton
2014 Pembroke Rd. ....Greensboro
Leinbach, Mrs. Lawrence Brickenstein
260 Kenleigh Circle ....Winston-Salem
Lennon, Mrs. Hershel Clanton
911 Sunset Dr. ....Greensboro
Leonard, Mrs. Walter Evan
104 27th St., N.W. ....Hickory

Leonard, Mrs. William A., Jr.
209 W. Bessemer ....Greensboro
Levi, Mrs. George Albert
605 Pearl St. ....Fayetteville
Lewis, Mrs. Charles Pell, Jr.
813 S. Main ....Reidsville
Lewis, Mrs. Clifford Whitfield
322 Woodrow ....High Point
Lewis, Mrs. Dockery Durham, Jr.
808 Henkle Road ....Statesville
Lewis, Mrs. John Sumter
362 N. Center St. ....Hickory
Lewis, Mrs. Martin Thomas ....Beaufort
Lewis, Mrs. Robert Edward
Finley Park ....North Wilkesboro
Lide, Mrs. Thomas Norwood
601 Barnsdale Rd. ....Winston-Salem
Ligon, Mrs. Harold Belton
43 Beverly Apt. ....Asheville
Liles, Mrs. George Welch
Williamsburg Road ....Concord
Liles, Mrs. Lonnie Carl
3025 Randolph Dr. ....Raleigh
Lilly, Mrs. James M.
226 Bradford Rd. ....Fayetteville
Lilly, Mrs. William Harold
Benson Highway ....Dunn
Lindsay, Mrs. Robert Boyd
King's Mill Road ----------------Chapel Hill
Link, Mrs. Melvin Robert
1050 Ardsley Rd. ....Charlotte
Lippett, Mrs. Devereaux H.
2109 Woodland Ave. ....New Bern
Little, Mrs. Howard Q. L.
Box 205 ....Gibsonville
Little, Mrs. Joseph Rice
Oak Rd. ....Salisbury
Littlejohn, Mrs. James Talmadge
8 Cedarcliff Rd. ....Asheville
Littlejohn, Mrs. Thomas Willard
2402 Forest Dr. ....Winston-Salem
Littleton, Mrs. Leonidas Rosser, Jr. ....Mt. Airy
Liverman, Mrs. Henry Joseph ....Engelhard
Liverman, Mrs. Joseph Thomas ....Nashville
Llewellyn, Mrs. Charles Elroy, Jr.
3550 Hamstead Court ....Durham
Lock, Mrs. Frank Ray
1819 Buena Vista Rd. ....Winston-Salem
Lockhart, Mrs. David Armistead
Rt. 3, Burrage Rd. ....Concord
Lockhart, Mrs. Walter Samuel, Jr.
412 Carolina Circle ....Durham
Lodmell, Mrs. Elmer Arthur
1308 Cornwallis ....Greensboro
Lohr, Mrs. Dermot
Vance Circle ....Lexington
London, Mrs. Arthur Hill, Jr.
Shepherd and Wells Sts. ....Durham
Long, Mrs. Benjamin Leroy ....Glen Alpine
Long, Mrs. David Thomas
405 S. Main St. ....Roxboro
Long, Mrs. Glenn
630 N. Main St. ....Newton
Long, Mrs. Rowland V.
Erwin Heights ....Thomasville
Long, Mrs. Thomas Drumwright
513 S. Lamar St. ....Roxboro
Long, Mrs. Thomas Walter
N. Main St. ....Newton
Long, Mrs. Vann McKee
1021 West End Blvd. ....Winston-Salem
Long, Mrs. William Lunsford, Jr.
1103 Cowper Dr. ....Raleigh
Long, Mrs. William Matthews ....Mocksville
Long, Mrs. Zachary Filmore
214 Ann St. ....Rockingham
Longino, Mrs. Frank Henry
1914 Forest Hill Dr. ....Greenville
Lore, Mrs. Ralph Eli
407 Pennton Ave. ....Lenoir
Lott, Mrs. William Clifton
310 Vanderbilt Rd. ....Asheville
Lounsbury, Mrs. James Breckinridge
2519 Guilford Ave. ....Wilmington
Lovell, Mrs. William Figgatt
1517 Biltmore Dr. ....Charlotte
Lovill, Mrs. Robert Jones
Box 647 ....Mt. Airy
Lowery, Mrs. Charles D. ....Lowell
Lowery, Mrs. John Robert
1620 Wiltshire ....Salisbury
Lownes, Mrs. Milton Markley, Jr.
Redwheel Farm ....Dudley
Lucas, Mrs. Robert Theodore, Jr.
318 Chiswick Pl. ....Charlotte
Lumb, Mrs. George Dennett
1325 Hawthorne Road ....Wilmington
Lund, Mrs. Herbert Zachareus
3610 Kirby Dr. ....Greensboro
Lunsford, Mrs. Lewis, Jr.
20 Hy-Vu Drive ....Asheville
Lupton, Mrs. Carroll Crescent
3300 Starmount Dr. ....Greensboro
Lupton, Mrs. Emmett Stevenson
Box 485 ....Alamance
Lusk, Mrs. John A., III
1809½ Independence Rd. ....Greensboro
Lusk, Mrs. Walter Coles
1802 Independence Rd. ....Greensboro
Lutterloh, Mrs. Isaac Hayden, Sr.
202 McIver St. ....Sanford
Lutterloh, Mrs. Isaac Hayden, Jr.
510 Walnut Dr. ....Sanford
Lutz, Mrs. James Dwight
1125 Highland Ave. ....Hendersonville
Lyday, Mrs. Charles Emmett
819 S. York St. ....Gastonia
Lyday, Mrs. John E.
2205 Briarwood Dr. ....Greensboro
Lyday, Mrs. Russell Osborne
1610 Nottingham Rd. ....Greensboro
Lymberis, Mrs. Marvin Nicholas
2111 Radcliffe Ave. ... .Charlotte
Lynch, Mrs. John Franklin,.Jr.
905 Arbordale Dr. ....High Point

Lynn, Mrs. Cy Kellie
Bouchard St. ....Valdese
Lynn, Mrs. James Wiley, Jr.
Rockwood Acres ....Burlington
Lyon, Mrs. Brockton Reynolds
Country Club Apts. ....Greensboro

McAdams, Mrs. Charles Rupert, Sr.
31 W. Woodrow Ave. ....Belmont
McAdams, Mrs. Charles Rupert, Jr.
6331 Sardis Rd. ___ ________________Matthews
McAllister, Mrs. Hugh Alexander
Riverside Dr. ....Lumberton
McArn, Mrs. Hugh Munroe,
701 Anson Ave. ....Laurinburg
McBryde, Mrs. Angus Murdoch
3406 Westoner Rd. ....Durham
McCain, Mrs. John Lewis
1601 Highland ....Wilson
McCall, Mrs. Michael Alvin ....Marion
McCall, Mrs. William, Jr.
508 Walter Court ....Winston-Salem
McCallum, Mrs. James Archibald
75 Lee Drive ....Wilmington
McCampbell, Mrs. Leon C. ....Roanoke Rapids
McCarthy, Mrs. John Joseph
N. C. Sanatorium ....McCain
McCarty, Mrs. Ralph Leeves
843 Hempstead Pl. ....Charlotte
McCaskil, Mrs. Lloyd ....Maxton
McClees, Mrs. Edward Count ....Elm City
McClelland, Mrs. Joseph O. ....Maxton
McConnell, Mrs. Harvey Russell
1119 Cumberland Ave. ....Gastonia
McCoy, Mrs. Joseph Bennett, Jr.
2026 Sharon Lane ....Charlotte
McCracken, Mrs. Jesse William ..Guilford College
McCutcheon, Mrs. William B., Jr.
1429 Pennsylvania Ave. ....Durham
McDonald, Mrs. Con T.
1106 S. Madison Ave. ....Goldsboro
McDowell, Mrs. Harold Clyde
200 Arbor Rd. ....Winston-Salem
McDowell, Mrs. Roy Hendrix
20 Myrtle St. ....Belmont
McDuffie, Mrs. William Norman ....Robbins
McEachern, Mrs. Duncan Roland
1915 Hydrangea Pl. ....Wilmington
McElrath, Mrs. Percy John
2736 Toxey Dr. ....Raleigh
McFadyen, Mrs. Oscar Lee, Jr.
524 Valley Rd. ....Fayetteville
McGavran, Mrs. Edward G.
Greenwood Rd. ....Chapel Hill
McGee, Mrs. Julian Murrill
811 N. Elm St. ....Greensboro
McGill, Mrs. John Charles
506 Crescent Hill ....Kings Mountain
McGill, Mrs. Kenneth Harwood
505 Crescent Hill ....Kings Mountain
McGimsey, Mrs. James Franks, Jr.
Edgewood St. ....Morganton
McGowan, Mrs. Claudius ....Plymouth
McGowan, Mrs. Joseph Francis
303 Vanderbilt Rd. ....Asheville
McGrath, Mrs. Frank Bernard
212 E. 17th St. ....Lumberton
McGuffin, Mrs. William Christian
52 Forest Rd. ....Asheville
McIntosh, Mrs. Henry Deane
2406 N. Duke St. ....Durham
McKee, Mrs. John Sasser, Jr.
Broughton Hospital ....Morganton
McKee, Mrs. Lewis Middleton
3633 Hope Valley Rd. ....Durham
McKeel, Mrs. Millard Filmore
12 Bevlyn Dr. ....Asheville
McKenzie, Mrs. Wayland Nash
N. Tenth St. ....Albemarle
McKinnon, Mrs. George Edward
1836 Harris Road ....Charlotte
McKinnon, Mrs. William James
501 W. Wade St. ....Wadesboro
McLain, Mrs. Bill Reid
Box 328 ....Troutman
McLean, Mrs. Ewen Kenneth
1110 Queens Rd., W. ....Charlotte
McLean, Mrs. James Wilton
217 DeVane St. ....Fayetteville
McLendon, Mrs. Walter Jones
Box 116 ....Oakboro
McLeod, Mrs. John Calvin, Jr.
707 Pou St. ....Goldsboro
McLeod, Mrs. John Purl Uttley ....Marshville
McLeod, Mrs. William Leslie
1504 Biltmore Dr. ....Charlotte
McLeod, Mrs. William Louis
S. Main St. ....Norwood
McManus, Mrs. Hugh Forrest, Jr.
3331 White Oak Rd. ....Raleigh
McMillan, Mrs. James Fulford
907 Live Oak Pkwy. ....Wilmington
McMillan, Mrs. Robert Lindsay
718 Arbor Rd. ....Winston-Salem
McMillan, Mrs. Robert Monroe
Massachusetts Ave. Ext. ....Southern Pines
McMillan, Mrs. Roscoe Drake
414 S. Main St. ....Red Springs
McMillan, Mrs. Thomas Henry, Jr.
1412 Scott Court ....Charlotte
McMurry, Mrs. Avery Willis
106 Hillside Dr. ....Shelby
McNeill, Mrs. Claude Ackle, Jr.
121 Church St. ....Elkin
McNiel, Mrs. Thomas Lee
N. Brook St. ....Wilkesboro
McPheeters, Mrs. Samuel Brown
307 Linwood Ave. ....Goldsboro
McPherson, Mrs. Charles Wade
422 Fountain Pl. ....Burlington
McPherson, Mrs. Samuel Dace, Jr.
29 Oak Dr. ....Durham
McQueen, Mrs. Robert Bruce, Jr.
Box 529 ....Graham
McRae, Mrs. James Thomas ....Elkin

McRae, Mrs. Marvin Everett
121 Beverly Pl. .... Greensboro
McRee, Mrs. Jean Douglas
808 Runnymeade Rd. .... Raleigh
McWhorter, Mrs. Robert Ligon
Northeast Drive .... Concord
Mabe, Mrs. Paul Alexander
122 Penrose Dr. .... Reidsville
Mabry, Mrs. Edward Bloxton
1034 W. Front St. .... Burlington
MacAlpine, Mrs. Orville Duncan
Route 2 .... Chandler
Macatee, Mrs. George, Jr.
25 Inglewood Rd. .... Asheville
MacDonald, Mrs. J. Kingsley
3600 Barclay Downs Dr. .... Charlotte
MacKay, Mrs. James Calvin
1805 Grace St. .... Wilmington
Mackie, Mrs. George Carlyle
Box 927 .... Wake Forest
MacLauchlin, Mrs. William Thompson ....Conover
MacRae, Mrs. John Donald
2813 Syke Dr. .... Fayetteville
Maddrey, Mrs. Milner Crocker
610 Franklin St. .... Roanoke Rapids
Maher, Mrs. James A.
Overbrook Drive .... Goldsboro
Major, Mrs. Richard Smart
816 Fourth Ave., W. .... Hendersonville
Maness, Mrs. Archibald Kelly
1918 Granville Rd. .... Greensboro
Maness, Mrs. Paul Franklin
1010 Central Ave. .... Burlington
Mangum, Mrs. Carlyle Thomas, Jr.
Highland Drive .... Leaksville
Manly, Mrs. Isaac Vaughan
2515 Lakeview Dr. .... Raleigh
Manly, Mrs. James Hollowell, Jr.
2100 St. James Rd. .... Raleigh
Manning, Mrs. Isaac Hall, Jr.
3901 Hope Valley Rd. .... Durham
Marder, Mrs. Gerard
Bridle Path Trail .... Gastonia
Marks, Mrs. Edgar Seymour
1112 Hamel Rd. .... Greensboro
Marr, Mrs. James Tilden
1718 Virginia Rd. .... Winston-Salem
Marshall, Mrs. James Flournoy
341 Arbor Rd. .... Winston-Salem
Marshburn, Mrs. Elisha Thomas, Jr.
2962 Park Ave. .... Wilmington
Martin, Mrs. Benjamin Franklin
2540 Warwick Rd. .... Winston-Salem
Martin, Mrs. Dan Anderson
Sourwood Drive .... Chapel Hill
Martin, Mrs. James Alfred
1305 Walnut St. .... Lumberton
Martin, Mrs. James Franklin
734 Roslyn Rd. .... Winston-Salem
Martin, Mrs. Sidney Arnold
2711 Fairview Rd. .... Raleigh
Martin, Mrs. William Francis
1534 Queens Rd., W. .... Charlotte
Mason, Mrs. Lockert Bemiss
824 Country Club Rd. .... Wilmington
Mason, Mrs. Manly .... Newport
Mason, Mrs. Philip
808 Henkel Rd. .... Statesville
Massey, Mrs. Charles Caswell
1318 Carlton Ave. .... Charlotte
Matheson, Mrs. Robert Arthur
Drawer 608 .... Raeford
Mathews, Mrs. Robert William
311 Meadowbrook Terr. .... Greensboro
Mathieson, Mrs. Kenneth Marlin .... Pittsboro
Matthews, Mrs. Hugh Archie .... Canton
Matthews, Mrs. Roland Dellwood
147 Tarleton Ave. .... Burlington
Matthews, Mrs. Vann M.
3010 Central Ave. .... Charlotte
Matthews, Mrs. William Camp
645 Hempstead Pl. .... Charlotte
Matthews, Mrs. William Walter
Oakland Heights .... Leaksville
Maulden, Mrs. Paul Ranzo
204 William St. .... Kannapolis
Mauzy, Mrs. Charles Hampton, Jr.
1820 Greenbriar Rd. .... Winston-Salem
Maxwell, Mrs. Clarence Schuyler .... Beaufort
May, Mrs. Harvey Craig
1136 Berkeley Ave. .... Charlotte
May, Mrs. William Joseph
1824 Georgia Ave. .... Winston-Salem
Maybin, Mrs. Richard Madden
Drawer M .... Lawndale
Mayer, Mrs. Walter Brem
2828 St. Andrews Lane .... Charlotte
Maynard, Mrs. Eugene Vincent
P. O. Box 155 .... Elm City
Meadows, Mrs. Joseph Herman
108 Clyde Ave. .... Wilson
Means, Mrs. Robert Lee
3040 Kinnamon Rd. .... Winston-Salem
Mease, Mrs. Willis Eugene
Box 327 .... Richlands
Mebane, Mrs. Giles Yancey
Carr St. .... Mebane
Mebane, Mrs. William Carter, Jr.
4507 Wrightsville Ave. .... Wilmington
Medlin, Mrs. Charles Thomas
Box 267 .... Rural Hall
Mees, Mrs. Theodore Howell
Maxton Rd. .... Lumberton
Melero, Mrs. Andres Tarcisio
Newell Heights .... Roxboro
Melton, Mrs. Robert Allen
Route 3, Box 192, Pirate's Cove ......Wilmington
Menefee, Mrs. Elijah Eugene, Jr.
2205 Cranford Rd. .... Durham
Menzies, Mrs. Henry Harding
814 Oaklawn Ave. .... Winston-Salem
Meritt, Mrs. Joseph E., Jr.
S-5 Raleigh Apts. .... Raleigh
Merritt, Mrs. Jesse Frederic
1615 S. College Park Dr. .... Greensboro

Merritt, Mrs. John Hamlett
Barnette Ave. .... Roxboro

Meschan, Mrs. Isadore
751 Roslyn Rd. .... Winston-Salem

Metcalf, Mrs. Lawrence Edward
Chunns Cove Rd. .... Asheville

Mewborn, Mrs. John Moses .... Farmville

Miller, Mrs. Andrew C., III
110 W. Mauney Circle .... Gastonia

Miller, Mrs. Cameron Eugene .... Jefferson

Miller, Mrs. Emery Clyde, Jr.
438 Lynn Ave. .... Winston-Salem

Miller, Mrs. George Rolfe
1040 Paramount Circle .... Gastonia

Miller, Mrs. Harry
108 Facility Dr. .... Fayetteville

Miller, Mrs. Henry Rankin
Fairway Drive .... Black Mountain

Miller, Mrs. Henry Shelton, Jr.
920 Goodwood Rd. .... Winston-Salem

Miller, Mrs. Ira Ben
1007 Westwood .... High Point

Miller, Mrs. Joseph Teles
914 Springdale Lane .... Gastonia

Miller, Mrs. Lloyd Davis .... Marion

Miller, Mrs. Oscar Lee
314 Fenton Place .... Charlotte

Miller, Mrs. Robert Carlysle
414 Harvie St. .... Gastonia

Miller, Mrs. Robert Evans
1101 Bolling Rd. .... Charlotte

Miller, Mrs. Robert Plato
P. O. Box 2017 .... Charlotte

Miller, Mrs. Walton Hoy, Jr.
1606 E. Mulberry St. .... Goldsboro

Miller, Mrs. Warren Edwin
502 Pinkney St. .... Whiteville

Milliken, Mrs. James Shepard
Box 55 .... Southern Pines

Milling, Mrs. James Reaves .... Waynesville

Miliman, Mrs. Theodore Harris
105 Glovinia St. .... Leaksville

Millns, Mrs. Dale Thomas
1316 National Ave. .... New Bern

Mills, Mrs. Wardell Hardee
1202 Country Club Dr. .... Greensboro

Minges, Mrs. Ray Donald
Longmeadow Rd. .... Greenville

Minick, Mrs. James Elder
E. Main St. .... Booneville

Mitchell, Mrs. Fred Neal
439-B Wakefield Dr. .... Charlotte

Mitchell, Mrs. George William
807 W. Kenan St. .... Wilson

Mitchell, Mrs. Roy Colonel .... Mt. Airy

Mitchell, Mrs. Thomas Brice
921 E. Marion .... Shelby

Mitchener, Mrs. Calvin Chambers
4865 Stafford Cl. .... Charlotte

Mitchener, Mrs. James Samuel, Jr.
Morrison Lane .... Laurinburg

Mock, Mrs. Charles Glenn
117 Greylyn Dr. .... Charlotte

Moffett, Mrs. Alexander Stuart
Alexander County Hospital .... Taylorsville

Mohr, Mrs. Jack Elmer
207 E. 17th .... Lumberton

Monroe, Mrs. Clement Rosenburg
Thayer Cottage .... Pinehurst

Monroe, Mrs. Daniel Geddie
204 Churchill Dr. .... Fayetteville

Monroe, Mrs. Edwin Wall
215 Library St. .... Greenville

Monroe, Mrs. John Howard
2642 Philip St. .... Winston-Salem

Monroe, Mrs. Lance Truman
Burrage Rd. .... Concord

Montgomery, Mrs. John Christian, Sr.
1532 Queens Rd. .... Charlotte

Montgomery, Mrs. John Christian, Jr.
2017 Radcliffe Ave. .... Charlotte

Montgomery, Mrs. Wayne Swope
55 Sunset Parkway .... Asheville

Moon, Mrs. Richard Young
49 Plymouth Circle .... Asheville

Moore, Mrs. Burmah Dixon
McAdenville Road .... Mount Holly

Moore, Mrs. Davis Lee
503 E. 5th St. .... Greenville

Moore, Mrs. D. Forrest
Box 136 .... Shelby

Moore, Mrs. Edward Eugene
32 Fairway Rd. .... Asheville

Moore, Mrs. Horace Greeley, Jr.
2905 Harvard Dr. .... Wilmington

Moore, Mrs. James LeGrant
2513 Colton Place .... Raleigh

Moore, Mrs. John Andrew
1513 Independence Rd. .... Greensboro

Moore, Mrs. Julian Alison
34 Hilltop Rd. .... Asheville

Moore, Mrs. Laurie Walker .... Beaufort

Moore, Mrs. Ralph Bryan
1339 Hawthorne Rd. .... Wilmington

Moore, Mrs. Robert Alexander
2415 Warwick Rd. .... Winston-Salem

Moore, Mrs. Robert Alexander, Jr.
605 Jennings Drive .... Wilmington

Moore, Mrs. Robert Ashe
1734 Queens Rd., W. .... Charlotte

Moore, Mrs. Robert Love
311 W. Washington St. .... Bessemer City

Moore, Mrs. William Locke
616 Myers Lane .... Greensboro

Moorefield, Mrs. Robert Hoyle
P. O. Box 931 .... Kannapolis

Mordecai, Mrs. Alfred
806 S. Hawthorne Rd. .... Winston-Salem

Morehead, Mrs. Robert Page
1051 Arbor Rd. .... Winston-Salem

Morey, Mrs. Milton B. .... Morehead City

Morgan, Mrs. Benjamin Edward
1205 Alta Vista Lane .... Rocky Mount

Morgan, Mrs. Burnice Earl
2 Cedarcliff Rd. .... Asheville

Morgan, Mrs. Charles Hermann
1408 S. York St. .... Gastonia
Morgan, Mrs. Grady Alexander
1 Cambridge Rd. .... Asheville
Moricle, Mrs. Charles Hunter
Crescent Drive .... Reidsville
Morris, Mrs. Donald Shonk
2398 Warwick Rd. .... Winston-Salem
Morris, Mrs. James Francis
803 S. Madison Ave. .... Goldsboro
Morris, Mrs. John Watson .... Morehead City
Morris, Mrs. Leslie Morgan
1122 S. Edgemont Ave. .... Gastonia
Morris, Mrs. Marshal Glenn, Jr.
3700 Starmount Dr. .... Greensboro
Morris, Mrs. Rae Henderson
67 Louise Ave. .... Concord
Morrison, Mrs. Frank .... Waynesville
Morrison, Mrs. Robert Holcombe
331 Fairfield Rd. .... Fayetteville
Morrison, Mrs. Roger William
65 Sunset Parkway .... Asheville
Morton, Mrs. Leslie B.
Jefferson Drive .... Greenville
Morton, Mrs. L. Thomas
3812 Abingdon Rd. .... Charlotte
Moss, Mrs. Paul .... Hudson
Muirhead, Mrs. Samuel John
Veterans Hospital .... Salisbury
Mullen, Mrs. Malcolm Preston
1813 W. Nash St. .... Wilson
Mundorf, Mrs. George
1350 Dresden Dr. .... Charlotte
Murchison, Mrs. David Reid
315 S. Third St. .... Wilmington
Murphy, Mrs. Gibbons Westbrook
22 Hampstead Rd. .... Asheville
Murphy, Mrs. Thomas Lynch
409 Mocksville Ave. .... Salisbury
Myers, Mrs. Richard Thomas
600 Kingsbury Circle .... Winston-Salem

Nailling, Mrs. Richard Cabot
85 St. Dunstans Rd. .... Asheville
Nalle, Mrs. Brodie C., Sr.
906 S. College St. .... Charlotte
Nance, Mrs. Charles Lee
1825 E. 7th St. .... Charlotte
Nance, Mrs. Frederick Lee, Jr.
Route 3, Box 130M .... Kannapolis
Nance, Mrs. John Wesley
410 Powell St. .... Clinton
Nanzetta, Mrs. Leonard Anes
2756 Windsor Rd. .... Winston-Salem
Nash, Mrs. Thomas Palmer, III
306 E. Colonial .... Elizabeth City
Naumoff, Mrs. Phillip
2320 Croydon Rd. .... Charlotte
Neal, Mrs. Charles B., III
3806 Hillgrand Cr. .... Durham
Neal, Mrs. John William
Main Street .... Gibson
Neal, Mrs. Joseph Walter
1344 Brooks Ave. .... Raleigh
Neal, Mrs. Rutherford Douglas
2532 Hampton Ave. .... Charlotte
Neblett, Mrs. Herbert C.
1111 Granville Rd. .... Charlotte
Neeland, Mrs. Eugene Crawford
1506 Grove St. .... Wilson
Neese, Mrs. Kenneth Earl
611 Lancaster Ave. .... Monroe
Nelson, Mrs. Charlotte
2205 Woodview Rd. .... Kinston
Nelson, Mrs. Sully .... Ayden
Nelson, Mrs. William Howell
Box 328 .... Clinton
Neville, Mrs .Cecil Howell .... Scotland Neck
Newell, Mrs. Ernest T.
Box 37 .... Dobson
Newman, Mrs. Glenn Carraway
Coharie Dr. .... Clinton
Newman, Mrs. Harold Hastings, Jr.
11 Oak Rd. .... Salisbury
Newsome, Mrs. Henry Clay
Box 385 .... Pilot Mountain
Newton, Mrs. Howard Lowell
244 Hempstead Pl. .... Charlotte
Newton, Mrs. William King
Finley Park .... North Wilkesboro
Niblock, Mrs. Franklin Chalmers, Jr.
136 S. Union St. .... Concord
Nichols, Mrs. Austin Flint
122 Academy St. .... Roxboro
Nichols, Mrs. Rhodes Edmond, Jr.
1626 University Dr. .... Durham
Nichols, Mrs. Thomas Rogers
306 W. Union St. .... Morganton
Nicholson, Mrs. Henry Hale, Jr.
635 Manning Drive .... Charlotte
Nicholson, Mrs. William McNeal
824 Anderson St. .... Durham
Nifong, Mrs. Frank Miller .... Clemmons
Noble, Mrs. Baxter G.
604 Rudolph .... Goldsoboro
Noel, Mrs. George Thompson
P. O. Box 397 .... Kannapolis
Nolan, Mrs. James Onslow
P. O. Box 813 .... Kannapolis
Noland, Mrs. Robert Earl
785 Arbor Rd. .... Winston-Salem
Norfleet, Mrs. Charles Millner, Jr.
2566 Warwick Rd. .... Winston-Salem
Norment, Mrs. William Blount
702 Woodland Dr. .... Greensboro
Norris, Mrs. Louis Jerome, Jr. .... Morehead City
North, Mrs. Ellsworth Howard, Jr.
Riverview Crescent .... Elizabeth City
Norton, Mrs. Howard Binning
Route 1, Mills River Valley .... Horse Shoe
Norton, Mrs. John W. Roy
2129 Cowper Dr. .... Raleigh
Nowlan, Mrs. Fagg Bernard
P. O. Box 458 .... Pleasant Garden
Nowlin, Mrs. George Preston
946 Bromley Rd. .... Charlotte

O'Brien, Mrs. Paul Stevens
2622 Bucknell Ave. ....Charlotte
Odom, Mrs. Guy Leary
2812 Chelsea Cl., Hope Valley ....Durham
Odom, Mrs. Robert Edwin
99 Evelyn Place ....Asheville
Odom, Mrs. Robert Taft
1809 Virginia Rd. ....Winston-Salem
Oehlbeck, Mrs. Luther William F., Sr.
618 Third Ave., N. W. ....Hickory
Oehlbeck, Mrs. Luther William F., Jr.
Tremont Park ....Lenoir
Oelrich, Mrs. August M.
613 Palmer Dr. ....Sanford
Offutt, Mrs. Vernon Delmus
910 Rountree St. ....Kinston
Ogburn, Mrs. Herbert Hammond
1806 W. Market ....Greensboro
Ogburn, Mrs. Leon N.
1623 Canterbury Rd. ....Raleigh
Ogburn, Mrs. Lundie Calvin
945 Kenleigh Cl. ....Winston-Salem
Oleen, Mrs. George Gerhard
Medlin Rd. ....Monroe
Olive, Mrs. Percy Wingate
1322 Woodland Dr. ....Fayetteville
Oliver, Mrs. Jim Upton
2624 Fairview Rd. ....Raleigh
Oliver, Mrs. Joseph Andrew
Box 458 ....Rockwell
O'Quinn, Mrs. Edward Nelson
1810 Princess St. ....Wilmington
Ormand, Mrs. John William
309 Lancaster Ave. ....Monroe
Ormond, Mrs. Allison Lee
108 Sixth Ave., N. W. ....Hickory
Outlaw, Mrs. Jackson Kent
808 Pee Dee Ave. ....Albemarle
Owen, Mrs. Duncan Shaw
201 Oakridge Ave. ....Fayetteville
Owen, Mrs. John Fletcher
2631 Fairview Rd. ....Raleigh
Owen, Mrs. William Boyd ....Waynesville
Owens, Mrs. Francis Leroy
Linden Road ....Pinehurst
Owens, Mrs. Zack Doxey
Taylor's Beach ....Camden
Owsley, Mrs. Lawrence Hayes
Cove Canem Drive ....Boone

Pace, Mrs. Karl Busbee
404 Summit St. ....Greenville
Pace, Mrs. Samuel Eugene
Rt. 2, Box 52 ....Wilmington
Packard, Mrs. Douglas Richards
P. O. Box 22 ....Clinton
Padgett, Mrs. Charles King
Cleveland Springs ....Shelby
Padgett, Mrs. Philip Grover
605 N. Piedmont Ave. ....Kings Mountain
Page, Mrs. Ernest Benjamin, Jr.
2207 Wheeler Rd. ....Raleigh
Page, Mrs. George Dantzler
1855 Cassamia Pl. ....Charlotte

Painter, Mrs. William Watson
920 N. Main St. ....Mooresville
Palmer, Mrs. Yates Shuford
Louise Rd. ....Valdese
Palmes, Mrs. Wesley Calhoun, Jr.
440 Ridgeway Ave. ....Statesville
Papineau, Mrs. Alban ....Plymouth
Parham, Mrs. Asa Richmond
1045 Rockford Rd. ....High Point
Parker, Mrs. Charles Council
114 Warren ....Wilson
Parker, Mrs. John Wesley, Jr. ....Seaboard
Parker, Mrs. Oscar Lee
706 College St. ....Clinton
Parker, Mrs. Roy Turnage
111 Pinecrest Rd. ....Durham
Parker, Mrs. Samuel Lester, Jr.
1202 Harding Ave. ....Kinston
Parker, Mrs. Shepherd Falkener
Cleveland Springs ....Shelby
Parker, Mrs. Talbot Fort, Jr.
603 Prince Ave. ....Goldsboro
Parkinson, Mrs. Thomas William
417 Thomas Trail ....Gastonia
Parks, Mrs. William Craig
Emerywood Estates ....High Point
Parrish, Mrs. Alva E.
1317 Drumcliff Rd. ....Winston-Salem
Parrott, Mrs. Frank Strong
322 Mocksville Ave. ....Salisbury
Parrott, Mrs. John Arendall
2206 Woodview Rd. ....Kinston
Parsons, Mrs. Lacy Jack, Jr.
2404 Rowland Ave. ....Lumberton
Parsons, Mrs. William Herbert ....Ellerbe
Partrick, Mrs. Cornelius T.
Shady Banks ....Washington
Paschal, Mrs. George Washington, Jr.
3334 Alamance Dr. ....Raleigh
Paschold, Mrs. John Henry
Park Lane ....Albemarle
Pate, Mrs. Archibald Hanes
110 S. Oleander Ave. ....Goldsboro
Pate, Mrs. James Lloyd ....Fairmont
Pate, Mrs. William Henry ....Pikeville
Patrick, Mrs. Simmons Isler
2202 Greenbriar Rd. ....Kinston

Patterson, Mrs. Carl Norris
3930 Plymouth Rd., Hope Valley ....Durham

Patterson, Mrs. F. M. Simmons
1507 Tryon Rd. ....New Bern

Patterson, Mrs. Fred Geer
511 Senlac Rd. ....Chapel Hill

Patterson, Mrs. Hubert Clifton
Pittsboro Rd. ....Chapel Hill

Patterson, Mrs. Thomas ....Farmville

Patton, Mrs. John Donald
56 Elk Mtn. Scenic Hwy. ....Asheville

Patton, Mrs. William Hugh, Jr.
109 Terrace Pl. ....Morganton

Payne, Mrs. Clifton G.
1203 Morgan Drive ....Reidsville

Peak, Mrs. Latham Conrad
409 Lafayette St. .... Clinton
Pearse, Mrs. Richard Lehmer
713 Anderson St. .... Durham
Pearson, Mrs. John Kent
Pearson St. .... Apex
Peck, Mrs. Harold Artemus
425 Dogwood Lane .... Southern Pines
Peele, Mrs. James Clarendon
1208 Perry Park Dr. .... Kinston
Peeler, Mrs. Forrest Edwards .... Maiden
Peete, Mrs. Charles Henry
2027 Woodrow St. .... Durham
Pender, Mrs. John Robert, III
701 Ashworth Rd. .... Charlotte
Penick, Mrs. George Dial
215 Hillcrest Rd. .... Raleigh
Pennington, Mrs. Glenn Walton
2200 Queens Road East .... Charlotte
Perrin, Mrs. Thomas Samuel, Jr.
1761 Sterling Rd. .... Charlotte
Perritt, Mrs. John Olin
1327 Hawthorne Rd. .... Wilmington
Perry, Mrs. David Russell
1120 Iredell St. .... Durham
Perry, Mrs. David Russell, Jr.
746 Sylvan Rd. .... Winston-Salem
Perry, Mrs. Glenn Grey
702 Sunset Dr. .... High Point
Perry, Mrs. Henry Baker, Jr.
208 Homewood Dr. .... Greensboro
Perry, Mrs. Solomon Paul
3602 Rugby Rd., Hope Valley .... Durham
Perryman, Mrs. Olin Charles, Jr.
3312 Anderson Dr. .... Winston-Salem
Persons, Mrs. Elbert Lapsley
732 Anderson St. .... Durham
Peters, Mrs. August Richard, Jr.
Washington Park .... Washington
Peters, Mrs. William Anthony, Jr.
206 S. Road .... Elizabeth City
Pettus, Mrs. William Henry, Jr.
2051 Cassamia Pl. .... Charlotte
Pfeiffer, Mrs. John B., Jr.
Rugby Rd. .... Durham
Phifer, Mrs. William Houston
Lancaster Rd. .... Monroe
Phillips, Mrs. Charles A. Speas
525 E. Massachusetts Ave. .... Southern Pines
Philips, Mrs. Charles Kenneth
Box 8 .... Skyland
Phillips, Mrs. Ernest Nicholas
Finley Park .... North Wilkesboro
Phillips, Mrs. Marvin W.
Erwin Heights .... Thomasville
Phillips, Mrs. William Allen
Route 2, Greenville Sound .... Wilmington
Pickard, Mrs. Henry Mack
5002 Oleander Dr. .... Wilmington
Pickrell, Mrs. Kenneth L.
3 Sylvan Rd. .... Durham
Pierce, Mrs. Edwin
824 Chamberlain St. .... Raleigh
Pigford, Mrs. Robert Toms ....
155 Colonial Dr. .... Wilmington
Pishko, Mrs. Michael Thomas
Midland Rd. .... Pinehurst
Pittman, Mrs. Alfred Roland, Jr.
2304 Rowland Ave. .... Lumberton
Pittman, Mrs. Dorn Carl
Alamance Acres .... Burlington
Pittman, Mrs. Malory Alfred
905 Raleigh Rd. .... Wilson
Pittman, Mrs. William Austin
118 Stedman Ave .... Fayetteville
Pitts, Mrs. William Reid
429 Eastover Rd. .... Charlotte
Piver, Mrs. James DeCamp
Bayview Blvd. .... Jacksonville
Piver, Mrs. William Crawford, Jr.
Washington Park .... Washington
Pixley, Mrs. Roland Theo
2018 Bucknell .... Charlotte
Plonk, Mrs. George Webb
Crescent Hill .... Kings Mountain
Plyler, Mrs. Cranford O., Jr.
504 Pontiac Dr. .... Thomasville
Podger, Mrs. Kenneth Arthur
217 E. Markham Ave. .... Durham
Pollock, Mrs. Frank Edward
2406 Elizabeth Ave. .... Winston-Salem
Pollock, Mrs. Raymond
204 Hillcrest Rd. .... New Bern
Pool, Mrs. Bennett Baucom
2301 Buena Vista Rd. .... Winston-Salem
Poole, Mrs. Robert Franklin, Jr.
1631 St. Mary's St. .... Raleigh
Pope, Mrs. Henry T.
304 E. 17th St. .... Lumberton
Pope, Mrs. Robert Clyde
404 Monticello Dr. .... Wilson
Porter, Mrs. Richard Allison
Haywood Forest .... Hendersonville
Poteat, Mrs. Hubert McNeill, Jr.
422 Church St. .... Smithfield
Pott, Mrs. Walter Hawks
102 Lakewood Dr. .... Greenville
Powell, Mrs. Albert Henry
1632 University Dr. .... Durham
Powell, Mrs. Eppie Charles, Jr.
804 E. Park Ave. .... Goldsboro
Powell, Mrs. Jack
1951 Haywood Rd. .... Asheville
Powell, Mrs. John D. .... Mount Airy
Powers, Mrs. Frank Poydras
2529 White Oak Rd. .... Raleigh
Powers, Mrs. John Alfred
2035 Sherwood Rd. .... Charlotte
Prather, Mrs. Fonzo Goff
131 Cambridge Rd. .... Asheville
Prefontaine, Mrs. Joseph Edouard
901 Dover Rd. .... Greensboro
Pressly, Mrs. Claude Lowry
1863 Cassamia Pl. .... Charlotte
Pressly, Mrs. David Lowry
576 Dogwood Rd. .... Statesville

Prevette, Mrs. John Edgar
514 S. First St. ....Smithfield
Price, Mrs. Harry Johnson
2830 Glenn Ave. ....Winston-Salem
Prince, Mrs. George Edward
807 Townsend Ave. ....Gastonia
Printz, Mrs. Don Ralph
340 Midland Dr. ....Asheville
Pritchard, Mrs. George Littleton
119 Church St. ....Black Mountain
Pritchett, Mrs. Newton George
1705 St. Mary's St. ....Raleigh
Proctor, Mrs. James Thornton
428 Ridgefield Rd. ....Chapel Hill
Proctor, Mrs. Richard Culpepper
381 Westview Dr. ....Winston-Salem
Pugh, Mrs. Charles Harrison
610 S. Lee St. ....Gastonia
Pugh, Mrs. Raeford Theordore
Shady Banks ....Washington
Pugh, Mrs. Steve C. ....Chadbourn
Pugh, Mrs. Vernon Watson
1618 Oberlin Rd. ....Raleigh
Pulliam, Mrs. Benjamin Eloth
Robin Hood Rd. ....Winston-Salem
Pumphrey, Mrs. Albert Franklin
Box 627 ....Elizabethtown
Putney, Mrs. Robert Hubbard, Jr. ....Elm City

Queen, Mrs. Hugh Oscar
Rollins Ave. ....Hamlet
Query, Mrs. Robert Zimri, Jr.
1901 Matheson Ave. ....Charlotte
Quickel, Mrs. John Cephas
1140 S. Edgemont Ave. ....Gastonia
Quinn, Mrs. Clifton Lee ....LaGrange

Rabil, Mrs. William Edmond
1775 Buena Vista Rd. ....Winston-Salem
Rabold, Mrs. Bernard Louis
Dogwood Hills ....Newton
Rabold, Mrs. Leonard James
109 W. Newlyn St. ....Greensboro
Raby, Mrs. William Thomas
212 Bucknell Ave. ....Charlotte
Rachlin, Mrs. Stanton A.
Veteran's Hospital ....Fayetteville
Raiford, Mrs. Fletcher Lindsay
Haywood Forest ....Hendersonville
Raiford, Mrs. Theodore Sidney
30 Cedarcliff Rd. ....Asheville
Rainey, Mrs. William Thomas, Sr.
1401 Ft. Bragg Rd. ....Fayetteville
Ramsaur, Mrs. Jackson Townsend
1011 Fairfield Dr. ....Gastonia
Rand, Mrs. Cecil Holmes ....Fremont
Raney, Mrs. Richard Beverly
Farrington Rd. ....Chapel Hill
Rankin, Mrs. Pressley Robinson, Jr. ....Ellerbe
217 Circle Dr. ....Concord
Rankin, Mrs. Richard Brandon, Sr.
33 Marsh St. ....Concord
Rankin, Mrs. Richard Brandon, Jr.
217 Circle Dr. ....Concord
Rankin, Mrs. Richard Eugene
Mt. Holly-Belmont Rd. ....Mt. Holly
Rankin, Mrs. Rufus Pinkney, Jr.
622 Ashworth ....Charlotte
Ranson, Mrs. John Lester, Sr.
620 Hermitage Ct. ....Charlotte
Ranson, Mrs. John Lester, Jr.
2819 Glendale Rd. ....Charlotte
Ranson, Mrs. William Alexander
1352 Harding Pl. ....Charlotte
Raper, Mrs. James Sidney
24 Cedarcliff Rd. ....Asheville
Rapp, Mrs. Ira Hammes
1922 Beverly Dr. ....Charlotte
Rasberry, Mrs. Edwin Albert, Jr.
200 S. Deans St. ....Wilson
Rathbun, Mrs. Lewis Standish
46 Forest Rd. ....Asheville
Ravenel, Mrs. Samuel Fitzsimons
106 Fisher Park Circle ....Greensboro
Ray, Mrs. Frank L.
2021 Dilworth Rd., W. ....Charlotte
Ray, Mrs. Ritz Clyde ....West Jefferson
Rayle, Mrs. Wiley Wallace ....Maiden
Reece, Mrs. John Cochrane
Riverside Dr. ....Morganton
Reece, Mrs. John David
206 East North ....Albemarle
Reeser, Mrs. Archibald Willard
108 Glovenia St. ....Leaksville
Reeves, Mrs. Robert James
920 Anderson St. ....Durham
Register, Mrs. John Francis
301 Rockford Rd. ....Greensboro
Reid, Mrs. Charles Hamilton, Jr.
324 Banbury Rd. ....Winston-Salem
Reid, Mrs. James William ....Lowell
Reid, Mrs. Ralph Conner ....Pineville
Reid, Mrs. Robert Leary
646 W. Park Dr. ....Lincolnton
Reid, Mrs. William Joseph
2301 Danbury Rd. ....Greensboro
Reinhardt, Mrs. James Franklin
803 Starmount Dr. ....Durham
Rendleman, Mrs. David Atwell
1015 Holmes St. ....Salisbury
Reynolds, Mrs. Ernest
Morgan Dr. ....Reidsville
Reynolds, Mrs. Frank Russell
1210 Fairway Dr. ....Wilmington
Rhodes, Mrs. James Kent
3350 Alamance Dr. ....Raleigh
Rhodes, Mrs. John Sloan
2704 Vanderbilt Ave. ....Raleigh
Rhyne, Mrs. Sam Albertus
632 Greenway Dr. ....Statesville
Ribet, Mrs. James Ernest ....Rutherford College
Rice, Mrs. A. Douglas
1515 Ruffin St. ....Durham
Rice, Mrs. Donald Harpster
704 S. Hayne St. ....Monroe
Rice, Mrs. William T.
318 Mocksville Ave. ....Salisbury

Richards, Mrs. Robert D. ....Rock Ridge
Richardson, Mrs. Ernest Christopher, Jr.
1606 Lucerne Way ....New Bern
Richardson, Mrs. George Irvin
S. Park Dr. ....Reidsville
Richardson, Mrs. James Justis
Prince St. ....Laurinburg
Richardson, Mrs. William Perry
Box 758 ....Chapel Hill
Richman, Mrs. Samuel
3903 Madison Ave. ....Greensboro
Riddle, Mrs. Harry Duff
619 W. Hillcrest Ave. ....Gastonia
Ridge, Mrs. Clyde Franklin
609 Colonial Dr. ....High Point
Riggs, Mrs. Millard McAdoo
609 W. Union St. ....Morganton
Riley, Mrs. William Allen
617 Brent St. ....Winston-Salem
Riley, Mrs. William J.
222 W. Gate St. ....Newton
Rippy, Mrs. William Dennis
617 N. Sellars Mill Rd. ....Burlington
Roach, Mrs. Leonard Hunter
25 Sunset Parkway ....Asheville
Roach, Mrs. Robert Burchell
520 Westview St. ....Lenoir
Robbins, Mrs. Grover Jay
3718 Reynolda Rd. ....Winston-Salem
Robbins, Mrs. Jack Guyes
1408 Woodborn Rd. ....Durham
Roberson, Mrs. Robert Stuart ....Hazelwood
Roberts, Mrs. Bennet Watson
1503 W. Pettigrew St. ....Durham
Roberts, Mrs. Louis Carroll
3920 Plymouth Rd. ....Durham
Roberts, Mrs. Rufus Winston
2727 Canterbury Trail ....Winston-Salem
Roberts, Mrs. William McKinley
Babington Heights ....Gastonia
Robertson, Mrs. Carroll Bracey ....Jackson
Robertson, Mrs. Charles Gurney, Jr.
540 Country Club Dr. ....Mt. Airy
Robertson, Mrs. Edwin Mason
1934 Hermitage Ct. ....Durham
Robertson, Mrs. James Mebane ....Harmony
Robertson, Mrs. John Kenneth ....Pembroke
Robertson, Mrs. John Newton, Sr.
807 Hay St. ....Fayetteville
Robertson, Mrs. Leon Whitfield
401 Shady Circle Dr. ....Rocky Mount
Robertson, Mrs. Lloyd Harvey
4 North Road ....Salisbury
Robertson, Mrs. Logan Thomas
27 Fairmont Rd. ....Asheville
Robinson, Mrs. Charles Wilson
1114 Belgrave Pl. ....Charlotte
Robinson, Mrs. James Elbert
2701 Buena Vista Rd. ....Winston-Salem
Robinson, Mrs. James Thomas, Jr.
1305-D Eaton Pl. ....High Point
Robinson, Mrs. Joe
705 McDonald Ave. ....Hamlet

Rodler, Mrs. Dankwart E. H.
1312 National Ave. ....New Bern
Rodman, Mrs. Clark
Riverside ....Washington
Rodman, Mrs. Olzie Clark
519 W. Main St. ....Washington
Rogers, Mrs. Arthur Merriam
2115 Pinewood Cl. ....Charlotte
Rogers, Mrs. James Rufus
130 Hillsboro St. ....Raleigh
Rogers, Mrs. Max Pritchard
1112 Rolling Rd. ....High Point
Rogers, Mrs. Seymour Shulman
1503 Alandale Rd. ....Greensboro
Romeo, Mrs. Bruno Joseph
Laurel Park ....Hendersonville
Romm, Mrs. William Henry
Puddin' Ridge ....Moyock
Rose, Mrs. Abraham Hewitt, Jr.
723 Lake Boone Trail ....Raleigh
Rose, Mrs. Ira Woodall, Jr.
1319 Canterbury Rd. ....Raleigh
Rose, Mrs. James William ....Pikeville
Ross, Mrs. Donald MacConnell
510 Fountain Pl. ....Burlington
Ross, Mrs. Joseph Alderman
1005 Pee Dee Ave. ....Albemarle
Ross, Mrs. Otho Bescent, Sr.
2424 Selwyn Ave. ....Charlotte
Ross, Mrs. Otho Bescent, Jr.
680 Llewellyn Pl. ....Charlotte
Ross, Mrs. Thomas E.
107 Anson Ave. ....Rockingham
Ross, Mrs. Willis Richard
736 E. Oakwood Ave. ....Albemarle
Rosser, Mrs. John Hays
603 E. Front ....Statesville
Roth, Mrs. O. Ralph
2900 Idlewood Circle ....Charlotte
Rousseau, Mrs. James Parks
808 Oaklawn Ave. ....Winston-Salem
Rowe, Mrs. Charles Roy, Jr.
633 Margaret Rd. ....Statesville
Royal, Mrs. Benjamin Franklin ....Morehead City
Royal, Mrs. Donnie Martin
Box 156 ....Salemburg
Royster, Mrs. Chauncey Lake
2607 Fairview Rd. ....Raleigh
Royster, Mrs. James Dan
Box 68 ....Benson
Ruark, Mrs. Robert James
3132 Sussex Rd. ....Raleigh
Rubin, Mrs. Adrian Stevens
104 Nutbush Rd. ....Greensboro
Ruffin, Mrs. Julian Meade
816 Anderson St. ....Durham
Russell, Mrs. Phillip Everitt
4 Deerfield Rd. ....Asheville
Russell, Mrs. William Marler
1 Lone Pine Rd. ....Asheville
Ryburn, Mrs. Samuel Benjamin
1133 W. Nash St. ....Wilson

Sadler, Mrs. Ralph Colvert
106 S. Madison St. .... Whiteville
Saleeby, Mrs. Richard George, Jr.
2307 Churchill Rd. .... Raleigh
Salle, Mrs. George Fredric
Isabella Ave. .... Washington
Salter, Mrs. Theodore .... Beaufort
Salters, Mrs. Frederic Hay
1103 Riverside .... Elizabeth City
Sample, Mrs. Robert Cannon
Dana Rd. .... Hendersonville
Sanders, Mrs. Lee Hyman
2502 Anderson Dr. .... Raleigh
Sandy, Mrs. Robert E.
220 E. Main St. .... Washington
Sanger, Mrs. Paul Weldon
1813 Providence Rd. .... Charlotte
Santos, Mrs. Juan J.
212 Pennsylvania Ave. .... Winston-Salem
Sardi, Mrs. Carl Anthony
508 Willowbrook Dr. .... Greensboro
Sargeant, Mrs. Angus Gus
322 Otteray .... High Point
Sasser, Mrs. Patrick H.
108 S. Andrews Ave. ________ Goldsboro
Saunders, Mrs. Charles Lawrence, Jr.
523 Wildwood Lane .... Burlington
Saunders, Mrs. John Turner
29 Maywood Rd. ________ Asheville
Saunders, Mrs. Stanley Stewart
1322 Greenway Dr. ________ High Point
Savage, Mrs. Robert Thomas
385 Plymouth .... Winston-Salem
Sawyer, Mrs. Charles Glenn
812 Sylvan Rd. ________ Winston-Salem
Sawyer, Mrs. Logan Everett
Ashe St. .... Elizabeth City
Scarborough, Mrs. Charles Foster, Jr. ______ Star
Schafer, Mrs. Earl William
Emerywood Estates ________ High Point
Scherer, Mrs. Irvin George
Box 23 ________ Hamptonville
Schiebel, Mrs. Herman Max
1020 Anderson St. ________ Durham
Schlaseman, Mrs. Guy W.
3437 Rugby Road .... Durham
Schoenheit, Mrs. Edward William
25 Eastwood Rd. ________ Asheville
Schoonover, Mrs. R. A.
2107 Lafayette Ave. .... Greensboro
Schorr, Mrs. Robert
403 W. Church .... Elizabeth City
Schweizer, Mrs. Donald Conrad
903 Dover Rd. .... Greensboro
Scott, Mrs. Alan Fulton
Mocksville Rd. ________ Salisbury
Scott, Mrs. Peter Somers
Route 2 ________ Burlington
Scott, Mrs. Samuel Floyd
Route 2 ________ Burlington
Seagle, Mrs. Lee M., Jr.
125 1st Ave., S. E. .... Hickory

Seavy, Mrs. Paul W.
415 Carolina Circle ________ Durham
Seay, Mrs. Hillis Ledbetter
Auten .... Huntersville
Seear, Mrs. Torben
938 Paramount Circle ________ Gastonia
Selby, Mrs. William Elledge
1126 Belegrave Pl. ________ Charlotte
Semans, Mrs. James Hustead
1415 Bivins St. ________ Durham
Senior, Mrs. Robert Joseph
228 Hayes Rd. .... Chapel Hill
Senter, Mrs. William Jeffress
2330 Churchill Rd. ________ Raleigh
Sessions, Mrs. John Turner, Jr.
Morgan Creek Rd. ________ Chapel Hill
Setnor, Mrs. Stanford
220 Facility Dr. ________ Fayetteville
Severn, Mrs. Henry Doeller
4 Pine Tree Rd. ________ Asheville
Shackelford, Mrs. Robert Hilliard
201 W. Pollock St. ________ Mt. Olive
Shafer, Mrs. Irving Everett, Sr.
230 W. Thomas St. ________ Salisbury
Shafer, Mrs. Irving Everett, Jr.
618 Margaret Dr. ________ Statesville
Shaffner, Mrs. Louis deS.
818 Sylvan Rd. ________ Wiston-Salem
Shaia, Mrs. William Harry
2245 Mecklenburg ________ Charlotte
Shannon, Mrs. George Ward
Deweese Ave. ________ Rockingham
Sharp, Mrs. Oliver Ledbetter
214 Country Club Dr. .... Greensboro
Sharp, Mrs. William Thomas
Veterans Hospital ________ Salisbury
Sharpe, Mrs. Eugene Baxter
288 Kenilworth ________ Asheville
Sharpe, Mrs. Frank Alexander
111 E. Hendrix St. ________ Greensboro
Shaver, Mrs. William Trantham
1105 Pee Dee Ave. ________ Albermarle
Shaw, Mrs. John Alexander
5948 Bragg Blvd. ________ Fayetteville
Shaw, Mrs. Lloyd Roosevelt
222 N. Oak St. ________ Statesville

Shearin, Mrs. W. Thad, Jr.
1163 Carolina Ave., N. ________ Carolina Beach
Shelburne, Mrs. Palmer Augustine
2311 Princess Ann St. ________ Greensboro
Shelburne, Mrs. Robert C.
159 Lakeshore Dr. ________ Asheville
Sherrill, Mrs. Harry B.
11 Broad .... Swansboro
Sherrill, Mrs. John Franklin, Jr.
3326 Rugby Rd., Hope Valley ________ Durham
Shingleton, Mrs. William Warner
3866 Summerset Dr. ________ Durham
Shinn, Mrs. George Clyde ________ China Grove
Shipley, Mrs. John LeRoy
309 W. Church ________ Elizabeth City

Shirey, Mrs. John Luther
Leicester Rd., Route 4 .... Asheville
Shoemaker, Mrs. Carroll Clifton
Route 2 .... Burlington
Shook, Mrs. Earl Lester, Jr.
37 Gracelyn Rd. .... Asheville
Shuford, Mrs. Jacob Harrison
1007 14th Ave., N. W. .... Hickory
Shull, Mrs. William Henry
2830 Belvedere Ave. .... Charlotte
Sieker, Mrs. Herbert Otto
204 Forestwood Dr. .... Durham
Siewers, Mrs. Christian Fogle
1908 Winterlochen Rd. .... Fayetteville
Sikes, Mrs. Charles Henry
3930 Madison Ave. .... Greensboro
Sikes, Mrs. Walter Allen
State Hospital .... Raleigh
Silverthorne, Mrs. Ray G.
Charlotte St. .... Washington
Silverton, Mrs. George
502 W. 26th St. .... Lumberton
Simmons, Mrs. Alexander Wingate
604 Glenwood Ave. .... Burlington
Simmons, Mrs. J. Dale .... Mount Airy
Simons, Mrs. Charles Ernest
614 Raleigh Rd. .... Wilson
Simpson, Mrs. Paul Ervin
2612 Dover Rd. .... Raleigh
Simpson, Mrs. Thomas E.
Box 327 .... Walnut Cove
Simpson, Mrs. Thomas William
763 Barnsdale Rd. .... Winston-Salem
Sinclair, Mrs. Carter Ashton
353 8th St., N. W. .... Hickory
Sinclair, Mrs. Louis Gordon
3309 White Oak Rd. .... Raleigh
Sinclair, Mrs. Robey Thomas, Jr.
155 Renovah Circle .... Wilmington
Singletary, Mrs. George Currie
Box 246 .... Clarkton
Singletary, Mrs. Henry Pate
3438 Wilshire Blvd. .... Wilmington
Singletary, Mrs. William Vance
32 Beverly Drive .... Durham
Sink, Mrs. Charles Shelton
Sunset Dr. .... North Wilkesboro
Sinnett, Mrs. John Franklin
524 W. 8th St. .... Newton
Siske, Mrs. Grady Cornell .... Pleasant Garden
Skeen, Mrs. Leo Brown
812 N. Main St. .... Mooresville
Skinner, Mrs. Benjamin Smith
418 S. Duke St. .... Durham
Slagle, Mrs. Thomas Dick
Box 456 .... Sylva
Slate, Mrs. Francis Wesley
Box 407 .... Mocksville
Slate, Mrs. John Samuel
1215 W. Fourth St. .... Winston-Salem
Slate, Mrs. Joseph Esmond
1051 Rockford Rd. .... High Point
Slate, Mrs. Marvin Longworth
100 Brantley Circle .... High Point
Sledge, Mrs. John Burton
305 Forest Lane .... Charlotte
Sloan, Mrs. Allen Barry
745 N. Main St. .... Mooresville
Sloan, Mrs. David Bryan
1116 Magnolia Pl. .... Wilmington
Sloan, Mrs. Henry Lee, Jr.
154 Canterbury Dr. .... Charlotte
Sluder, Mrs. Fletcher Sumpter
Chunns Cove Rd. .... Asheville
Sluder, Mrs. Harold Miles
2245 Roswell Ave. .... Charlotte
Smart, Mrs. Gardner Ford
58 St. Dunstans Rd. .... Asheville
Smedberg, Mrs. George Andrew
517 Circle Drive .... Burlington
Smeltzer, Mrs. Dave Harvey
Route 4, Box 380-K .... Matthews
Smerznak, Mrs. John Joseph
209 E. Corban St. .... Concord
Smethie, Mrs. William Massie
831 Burns St. .... Wadesboro
Smith, Mrs. Albert Heyward, Jr. .... Waynesville
Smith, Mrs. Alick T.
5306 Dorchester Rd. .... Greensboro
Smith, Mrs. Anderson Jones .... Black Creek
Smith, Mrs. Claiborne Thweat
208 Hickory St. .... Rocky Mount
Smith, Mrs. Everette Duane
Box 1360 .... Candler
Smith, Mrs. Foyell Pennington
Carolina & Kildee .... Lexington
Smith, Mrs. Harold Benjamin
Finley Park .... North Wilkesboro
Smith, Mrs. J. Graham, Jr.
1118 Woodburn Rd. .... Durham
Smith, Mrs. J. Lawton
3805 Hillgrand Cr. .... Durham
Smith, Mrs. James Jefcoat
1204 E. 3rd St. .... Greenville
Smith, Mrs. James McNeill .... Rowland
Smith, Mrs. Jay Leland, Jr.
225 N. Rowan Ave. .... Spencer
Smith, Mrs. John Goodrich
200 Wildwood Ave. .... Rocky Mount
Smith, Mrs. Joseph
1303 E. 5th St. .... Greenville
Smith, Mrs. Joseph Pinkney
935 Paramount Circle .... Gastonia
Smith, Mrs. Opie Norris
107 W. Avondale .... Greensboro
Smith, Mrs. Richard B.
121 S. Caldwell St. .... Salisbury
Smith, Mrs. Roy Meadows
206 Homewood Dr. .... Greensboro
Smith, Mrs. Sidney
905 Williamson Dr. .... Raleigh
Smith, Mrs. Slade Alvah
308 N. Madison St. .... Whiteville
Smith, Mrs. William Alexander
2310 White Oak Rd. .... Raleigh
Smith, Mrs. William G., Sr.
224 Colonial Drive .... Thomasville

Smith, Mrs. William Mitchell
516 Grand Blvd. ....Boone
Snelling, Mrs. John McLucius
1036 Queens Rd., W. ....Charlotte
Snipes, Mrs. Richard Dean
312 Valley Rd. ....Fayetteville
Snow, Mrs. Leo Beman
140 Pearson Dr. ....Morganton
Sohmer, Mrs. Marcus Frank, Jr.
811 Arbor Rd. ....Winston-Salem
Sommerville, Mrs. Lewis Cass
Rt. 2, Box 323M ....Chandler
Sorrell, Mrs. Furman Yates ....Lilesville
Sowers, Mrs. Roy Gerold
Brinn Drive ....Sanford
Spaeth, Mrs. Walter
Winslow Acres ....Elizabeth City
Spargo, Mrs. John Prichard ....Cooleemee
Sparrow, Mrs. Harry Ward
108 Nutbush, East ....Greensboro
Spaugh, Mrs. Earle
150 McAlway ....Charlotte
Speas, Mrs. Dallas Cleaborn
2598 Reynolda Rd. ....Winston-Salem
Speas, Mrs. William Paul, Sr.
437 Springdale Ave. ....Winston-Salem
Speas, Mrs. William Paul, Jr.
2915 Country Club Rd. ....Winston-Salem
Spencer, Mrs. Allen
506 Grove St. ....Salisbury
Spencer, Mrs. Frederick Brunell, Jr.
117 Lilly Ave. ....Salisbury
Spencer, Mrs. William Gear, Jr.
301 West End Ave. ....Wilson
Spigner, Mrs. Prescott Bush
1107 Perry St. ....Kinston
Spikes, Mrs. Vera B.
2745 Dogwood Rd. ....Durham
Spillman, Mrs. Louis Cromwell, Jr.
Dodson Mill Rd. ....Pilot Mountain
Sprunt, Mrs. William Hutchinson, Jr.
1931 Virginia Rd. ....Winston-Salem
Spudis, Mrs. Edward Verhines
Apt. 9 Wake Forest College ....Winston-Salem
Spurr, Mrs. Charles
1845 Buena Vista Rd. ....Winston-Salem
Squires, Mrs. Claude Babbington
2128 Malvern Rd. ....Charlotte
Stafford, Mrs. William R., Jr.
3400 Lennox Court ....Greensboro
Stallard, Mrs. Sam Kane
Parkway Boulevard ....Reidsville
Stallings, Mrs. Davey Bingham
Route 1 ....Clemmons
Stallings, Mrs. T. Lacy
2404 White Oak Rd. ....Raleigh
Stanfield, Mrs. Elwin
516 Country Club Dr. ....Fayetteville
Stanfield, Mrs. Wiliam Wesly
S. Layton Ave. ....Dunn
Stanley, Mrs. Sherburn Moore ....Enka
Starling, Mrs. Charles Ray
2128 Andover Rd. ....Charlotte
Stanton, Mrs. Allie McLeod ....Plymouth
Starling, Mrs. Howard Montford
123 Pine Valley Rd. ....Winston-Salem
Starling, Mrs. Wyman Plato ....Roseboro
Steen, Mrs. Claude Earl
Route 2 ....Chandler
Stegall, Mrs. John Thomas
327 Oakwood Dr. ....Statesville
Steiger, Mrs. Howard Paul
1927 Sharon Lane ....Charlotte
Stephen, Mrs. Charles Ronald
1608 University Dr. ....Durham
Stephens, Mrs. Freeman Irby
54 Sunset Parkway ....Asheville
Stephens, Mrs. Richard Samuel
306 N. Ridge Dr. ....Kannapolis
Stephenson, Mrs. Bennett Edward ....Rich Square
Sternbergh, Mrs. Waldemar C. A.
1217 Belgrave Pl. ....Charlotte
Stevens, Mrs. Hamilton Wright, Jr.
90 Grovewood Rd. ....Asheville
Stevens, Mrs. Joseph Blackburn
202 Homewood Ave. ....Greensboro
Stewart, Mrs. Albert, Jr.
1507 Morganton Rd. ....Fayetteville
Stewart, Mrs. Daniel Niven, Jr.
925 4th Ave., N. W. ....Hickory
Stewart, Mrs. Francis Asbury
722 Quarterstaff Rd. ....Winston-Salem
Stewart, Mrs. John Reagan
515 Walnut St. ....Statesville
Stewart, Mrs. Roy Allen
422 W. 9th St. ....Newton
Stiff, Mrs. Audrey Olin
335 Bouchard St. ....Valdese
Stines, Mrs. Ernest Harrison ....Canton
Stirewalt, Mrs. Neale Summers
703 E. Lexington Ave. ....High Point
Stockdale, Mrs. Wayne Harrop
911 S. Third St. ....Smithfield
Stocker, Mrs. Frederick W.
1124 Forest Hills Blvd. ....Durham
Stockton, Mrs. Irving Richard
919 Tatum Dr. ....New Bern
Stone, Mrs. Leslie Ozburn
922 Sycamore St. ....Rocky Mount
Stone, Mrs. Marvin Lee
1605 Riviera Dr. ....Rocky Mount
Stovall, Mrs. Horace Henry
210 Homewood Dr. ....Greensboro
Stratton, Mrs. James David
2165 Sharon Lane ....Charlotte
Strawcutter, Mrs. Howard Elsworth
404 W. 25th St. ....Lumberton
Streeter, Mrs. Charles Truman
19 Warlick Street ....Jacksonville
Strickland, Mrs. William H.
1009 Fassifern Court ....Hendersonville
Stringfield, Mrs. James King ....Waynesville
Stringfield, Mrs. Thomas, Jr. ....Waynesville
Strong, Mrs. Leonell Clarence, Jr.
110 Hilltop Lane ....Lenoir

Strong, Mrs. William M.
224 East Boulevard .... Charlotte
Strosnider, Mrs. Charles Franklin
127 S. John St. .... Goldsboro
Stroupe, Mrs. Albertus Ulla, Jr.
157 Oakland Ave. .... Mount Holly
Stuckey, Mrs. Charles LeGrand
2219 Beverly Dr. .... Charlotte
Styron, Mrs. Charles Woodrow
920 Wiliamson Dr. .... Raleigh
Sugg, Mrs. William Cunnigham
355 Roslyn Road .... Winston-Salem
Suiter, Mrs. Thomas Bayton, Jr.
100 S. Taylor St. .... Rocky Mount
Suiter, Mrs. Wester Ghio
501 Sycamore St. .... Weldon
Summerlin, Mrs. Arthur Rogers
3407 Churchill Rd. .... Raleigh
Summerlin, Mrs. Harry
218 E. Church St. .... Laurinburg
Summerlin, Mrs. Robert L. .... Dublin
Summers, Mrs. John Dent
524 Sixth St., N. W. .... Hickory
Sumner, Mrs. Emmett Ashworth
502 Overbrook Dr. .... High Point
Sutter, Mrs. Renzo Humberto
401 Main St. .... Mt. Airy
Sutton, Mrs. Homer George, Jr.
3700 Reynolda Rd. .... Winston-Salem
Sutton, Mrs. William Wayne
305 Hamlet .... Carolina Beach
Swain, Mrs. Wingate E.
Washington Park .... Washington
Swan, Mrs. Bill Joe
895 Arbor Lane .... Concord
Swann, Mrs. Cecil Collins
21 Browntown Rd. .... Asheville
Sweaney, Mrs. Hunter McGuire
1007 Vickers Ave. .... Durham
Sweeney, Mrs. C. Leslie, Jr.
301 Northwood Drive .... Raleigh
Sweeney, Mrs. Edgar Chew
4020 Ridgecrest Ave. .... Charlotte
Sykes, Mrs. Charles Louis
205 Rawley Ave. .... Mt. Airy
Sykes, Mrs. Ralph Judson
205 Rawley Ave. .... Mt. Airy
Sykes, Mrs. Rufus Preston
Box 428 .... Asheboro

Taliaferro, Mrs. Richard McCulloch
2311 Lafayette Ave. .... Greensboro
Tally, Mrs. Bailey Thomas
N. Tenth St. .... Albemarle
Tannenbaum, Mrs. Abraham Jack
1301 Latham Rd. .... Greensboro
Tart, Mrs. James Milton, Jr.
564-A Wakefield Dr. .... Charlotte
Tate, Mrs. Allen Denny, Jr.
415 Pine St. .... Graham
Tatum, Mrs. Walter Low
607 Mitchell Ave. .... Salisbury
Tayloe, Mrs. David Thomas
709 W. Main St. .... Washington
Tayloe, Mrs. John Cotten
Short Drive .... Washington
Taylor, Mrs. Andrew DuVal
2610 Selwyn Ave. .... Charlotte
Taylor, Mrs. Frederick Harvey
3642 Park Rd. .... Charlotte
Taylor, Mrs. John Richard
Box 313 .... Enka
Taylor, Mrs. Thomas Jefferson
614 Franklin St. .... Roanoke Rapids
Taylor, Mrs. Vernon Williams, Jr.
815 N. Bridge St. .... Elkin
Taylor, Mrs. William Ivey, Jr.
Box 156 .... Wilmington
Temple, Mrs. Rufus Henry
307 Wilson Ave. .... Kinston
Templeton, Mrs. Ralph Gordon
206 W. College Ave. .... Lenoir
Tenney, Mrs. Luman Harris
Route 1 .... Arden
Terrell, Mrs. Thomas Eugene
514 Hayworth Circle .... High Point
Thomas, Mrs. David Pryse
Rt. 2, Box 158 .... Wilmington
Thomas, Mrs. James Valentine
149 Highland Drive .... Leaksville
Thomas, Mrs. Julius Graham
117 S. Tremont Dr. .... Greensboro
Thomas, Mrs. Walter Lee
3615 Dover Rd., Hope Valley .... Durham
Thomas, Mrs. William Ralph
Rt. 1, Box 82 .... Elizabeth City
Thompson, Mrs. Alexander Frank, Jr.
Rt. 3, Burrage Rd. .... Concord
Thompson, Mrs. B. E., Jr.
303 W. Park St. .... Cary
Thompson, Mrs. Charles Robert
315 Highland Ave. .... Lenoir
Thompson, Mrs. Clive Allen .... Sparta
Thompson, Mrs. Fred Arrowwood
303 Highland Ave. .... Lenoir
Thompson, Mrs. George Richard Cunliff
101 E. Renovah Cir. .... Wilmington
Thompson, Mrs. Sanford Webb, Jr.
Morehead City
Thompson, Mrs. Silas Raymond
240 Cherokee Rd. .... Charlotte
Thompson, Mrs. Williard Chandler, Jr.
311 Cherokee Pl. .... Charlotte
Thompson, Mrs. Winfield Lynn
1304 E. Mulberry .... Goldsboro
Thornhill, Mrs. Edwin Hale
2828 Lakeview Dr. .... Raleigh
Thornhill, Mrs. George Tudor, Jr.
3021 Granville Dr. .... Raleigh
Thorp, Mrs. Adam Tredwell
543 Avent St. .... Rocky Mount
Thurmond, Mrs. Jack Alfred
3615 Henshaw Dr. .... Charlotte
Thurston, Mrs. Thomas Gardiner
120 Confederate Ave. .... Salisbury
Thorne, Mrs. Edward Young Cox
105 West End Ave. .... Wilson

Thorne, Miss Silas Owens, Jr. ....Morehead City
Tice, Mrs. Walter Thomas
411 Hillcrest Dr. ....High Point
Tidler, Mrs. James
702 Forest Hills Dr. ....Wilmington
Timmerman, Mrs. William Bledsoe
1960 Queens Rd., W. ....Charlotte
Timmons, Mrs. Robert L.
Fern Lane ....Chapel Hill
Todd, Mrs. Lester Claire
1029 Granville Rd. ....Charlotte
Tomlin, Mrs. Edwin Merrill
58 LeCline Dr. ....Concord
Tomlinson, Mrs. Robert Lee
W. Nash Rd. ....Wilson
Townsend, Mrs. William Ball
2200 Pinewood Cl. ....Charlotte
Trachtenberg, Mrs. William
Hillcrest Dr. ....Goldsboro
Trado, Mrs. Charles E.
333 2nd St. Pl., N. W. ....Hickory
Trevathan, Mrs. Gordon Earl, Jr.
1908 Forest Hill Dr. ....Greenville
Trivette, Mrs. Parks Dewitt
547 3rd St., N. E. ....Hickory
Troutman, Mrs. Baxter Suttles
511 Mt. View ....Lenior
Troxler, Mrs. Eulyss Robert
2314 Princess Ann St. ....Greensboro
Truslow, Mrs. Roy Earl
1708 Penrose Drive ....Reidsville
Tucker, Mrs. George Franklin ....Zebulon
Turlington, Mrs. William Troy, Jr.
Woodland Dr. ....Jacksonville
Turner, Mrs. John ....Fair Bluff
Turrentine, Mrs. Kilby Pairo
809 Rountree St. ....Kinston
Tuttle, Mrs. James Gray
530 N. Fifth St. ....Albemarle
Tuttle, Mrs. Marler Slate
201 Prof. Bldg., Tuttlewood Dr. ....Kannapolis
Tuttle, Mrs. Reuben Gray
784 Stratford Rd. ....Winston-Salem
Tyndall, Mrs. Hubert Durwood
1304 Evergreen Ave. ....Goldsboro
Tyndall, Mrs. Robert Glenn
413 Harding Ave. ....Kinston
Tyner, Mrs. Hugh Edward
Pinewood Road ....Gastonia
Tyner, Mrs. Kenneth Vann
363 Springdale Ave. ....Winston-Salem
Tyor, Mr.s Malcolm
922 Green St. ....Durham
Tyson, Mrs. Thomas David, Jr.
1106 Ferndale Dr. ....High Point
Tyson, Mrs. Woodrow Wilson
1012 Wellington ....High Point

Umphlet, Mrs. Thomas Leonard
2519 White Oak Rd. ....Raleigh
Underwood, Mrs. Harry Burnham
125 N. Race St. ....Statesville

Valk, Mrs. Henry Lewis
2828 Club Park Rd. ....Winston-Salem
Valone, Mrs. James Austin
1528 Iredell Dr. ....Raleigh
Van Blaricom, Mrs. Lawrence Stickney
7 Busbee Rd. ....Asheville
Van Doren, Mrs. Peter
Sunset Drive ....Hendersonville
Van Hoy, Mrs. Joe Milton
2204 Crescent Ave. ....Charlotte
Vann, Mrs. Robert Lee
1928 Virginia Rd. ....Winston-Salem
Vanore, Mrs. Andrew Albert
Box 456 ....Robbins
Van Velsor, Mrs. Harry
1304 Churchill Dr. ....Wilmington
Vatz, Mrs. Benjamin
2526 Fernwood Dr. ....Greensboro
Vaughan, Mrs. Roland Harris
N. Broad St. ....Edenton
Vaughn, Mrs. James Donald ....Hazelwood
Veazey, Mrs. Alex Halloway
Rhododendron Dr. ....Hendersonville
Vennart, Mrs. George G.
Woodhaven Road ....Chapel Hill
Verdery, Mrs. William Carey
1428 Raeford Rd. ....Fayetteville
Verdone, Mrs. George Frederick
3800 Wendover Cl. ....Charlotte
Verhoeff, Mrs. Dirk ....Huntersville
Verner, Mrs. Hugh David
2116 Sherwood Ave. ....Charlotte
Verner, Mrs. John Victor, Jr.
2401 Cranford Rd. ....Durham
Vernon, Mrs. James Taylor
120 Woodland Dr. ....Morganton
Vernon, Mrs. James William
209 Valdese Ave. ....Morganton
Vernon, Mrs. William Chester, Jr.
177 Woodland Rd. ....Asheville
Vestal, Mrs. Tom A.
1222 Stockton Rd. ....Kinston
Vetter, Mrs. John Stanley
212 Richmond Ave. ....Rockingham
Vinson, Mrs. T. Chalmes ....Laurel Hill
Vitolo, Mrs. Ralph E.
307 Sherman Dr. ....Fayetteville
Vollmer, Mrs. Donald Henry
Route 2, Box 300 ....Asheville
Vreeland, Mrs. Walling Douglas, Jr.
Rt. 1, Darwick Road ....Winston-Salem

Wadsworth, Mrs. Harvey B.
515 Broad St. ....New Bern
Waggoner, Mrs. Lonnie Austin, Jr.
2549 Pinewood Rd. ....Gastonia
Wainer, Mrs. Howard S.
607 N. Elam Ave. ....Greensboro
Walker, Mrs. Archie DuVall, Jr.
Westover Heights ....Edenton
Walker, Mrs. Harry Gordon
Route 4 ....Statesville
Walker, Mrs. John Barrett, Jr.
1222 May Ct. ....Burlington
Walker, Mrs. Samuel Haywood
63 Buchanan Ave. ....Asheville

Walker, Mrs. Thomas English
1200 Greylyn Dr. .... Charlotte
Wall, Mrs. George Ritchie
N. Tenth St. .... Albemarle
Wall, Mrs. Roger Irving
2707 Cambridge Rd. .... Raleigh
Wall, Mrs. Roscoe LeGrand, Jr.
822 N. Pine Valley Rd. .... Winston-Salem
Wallace, Mrs. John Dixon, Jr.
2564 Fairfax Dr. .... Gastonia
Waller, Mrs. Louis Clinton
Route 2, Box 136-A .... Candler
Walters, Mrs. Hezekiah Grover, Jr.
214 Jefferson St. .... Whiteville
Walton, Mrs. Cyrus Leslie .... Glen Alpine
Walton, Mrs. James Carey
125 Maehill Dr. .... Lenior
Wannamaker, Mrs. Edward Jones, Jr.
Route 3, Box 250 .... Charlotte
Wansker, Mrs. Bernard Arthur
1524 Biltmore Dr. .... Charlotte
Ward, Mrs. Bennie Brooks
306 Adelaide Drive .... Wilmington
Ward, Mrs. Doctor Ernest, Jr.
2206 Barker .... Lumberton
Ward, Mrs. Ernest
1015 E. Broad St. .... Statesville
Ward, Mrs. Frank Pelouze
1105 Riverside Dr. .... Lumberton
Ward, Mrs. Ivie Alphonso
211 Church St. .... Hertford
Ward, Mrs. John Charles .... LaGrange
Ward, Mrs. Wallace Clyde
1429 Canterbury Rd. .... Raleigh
Ward, Mrs. William Titus
917 Williamson Dr. .... Raleigh
Warren, Mrs. Casper Carl, Jr.
410 S. Green St. .... Wadesboro
Warren, Mrs. Joseph Benjamin
207 Pine Rd. .... New Bern
Warren, Mrs. Julian Marion .... Spring Hope
Warren, Mrs. Robert Lee
510 W. Harnett St. .... Dunn
Warrick, Mrs. Luby Albert
Route 1 .... Goldsboro
Warshauer, Mrs. Albert David
218 Forest Hills Dr. .... Wilmington
Warshauer, Mrs. Samuel Edward
2943 Hydrangea Pl. .... Wilmington
Warwick, Mrs. Hight Claudius
2320 Kirkpatrick Pl. .... Greensboro
Wassink, Mrs. William Klein
Route #343 .... Shiloh
Watkins, Mrs. Carlton Gunter
1223 Marlewood Terrace .... Charlotte
Watkins, Mrs. William Merritt
1423 Arcadia St. .... Durham
Watson, Mrs. John William
107 Forest Rd. .... Oxford
Watson, Mrs. Robert A.
Box 847 .... Elon College
Watters, Mrs. Vernon Gregg, Jr.
204 Rockingha mRd. .... Rockingham
Watts, Mrs. Walter Moore
40 Canterbury Rd. .... Asheville
Way, Mrs. John Edward .... Beaufort
Wear, Mrs. John Edmund
Country Club .... Salisbury
Weatherly, Mrs. Carl Holmes
1603 Independence Rd. .... Greensboro
Weathers, Mrs. Bahnson
928 Monroe St. .... Roanoke Rapids
Weathers, Mrs. Bailey Graham .... Stanley
Weathers, Mrs. Harry Huntington
401 Roanoke Ave. .... Roanoke Rapids
Weaver, Mrs. Richard Gray
1244 Irving St. .... Winston-Salem
Webb, Mrs. Alexander, Jr.
1019 Cowper Dr. .... Raleigh
Weeks, Mrs. John Francis
1304 Rivershore Rd. .... Elizabeth City
Weeks, Mrs. Kenneth Durham
1014 West Haven Blvd. .... Rocky Mount
Weinel, Mrs. William Harvey
4014 Evergreen Road .... Wilmington
Welfare, Mrs. Charles Randall
2641 Reynolds Rd. ---------- Winston-Salem
Wellborn, Mrs. William Revere, Jr.
300 Avery Ave. .... Morganton
Wells, Mrs. Edwin Julius
2802 Oleander Drive .... Wilmington
Welton, Mrs. David Goe
1900 Beverly Dr. .... Charlotte
Wessell, Mrs. John Charles
1501 Market St. .... Wilmington
West, Mrs. Bryan Clinton
Perry Park Dr. .... Kinston
West, Mrs. Clifton Forest
Perry Park Dr. .... Kinston
Wester, Mrs. Thaddeus Bryan
508 W. 28th .... Lumberton
Westmoreland, Mrs. Joseph Robert .... Canton
Weyher, Mrs. John E., Jr.
Overbrook Drive .... Goldsboro
Whaley, Mrs. James Davant
605 Third Ave., N. W. .... Hickory
Wharton, Mrs. Charles Watson
201 Meadowbrook .... Smithfield
Whicker, Mrs. Guy Lorraine
Route 1, Box 20 .... Kannapolis
Whicker, Mrs. Max Evans
504 S. Franklin St. .... China Grove
Whisnant, Mrs. Albert Miller
Park Rd., Route 2 .... Charlotte
Whitaker, Mrs. Donald Nash
1425 Canterbury Rd. .... Raleigh
Whitaker, Mrs. Paul F.
1205 N. Queen St. .... Kinston
Whitaker, Mrs. Richard Harper
120 N. Cherry St. .... Kernersville
White, Mrs. Edward Russell, Jr.
2634 Reynolds Rd. ---------- Winston-Salem
White, Mrs. James Stark
1807 Efland Dr. .... Greensboro
White, Mrs. Philip Fletcher
Stanley Ave. .... Rockingham

Vhite, Mrs. Francis Willard Moody ....Halifax
Vhite, Mrs. Thomas Preston
714 N. Edgehill Rd. ....Charlotte
Vhite, Mrs. William Elliott
3936 Churchill Rd. ....Charlotte
Vhitehead, Mrs. Seba Loren
341 Vanderbilt Rd. ....Asheville
Vhitener, Mrs. Donald Leonard
433 Lynn Ave. ....Winston-Salem
Vhitesides, Mrs. Edward Steele
215 N. Highland St. ....Gastonia
Vhitesides, Mrs. William Carl, Jr.
1500 Coventry Rd. ....Charlotte
Vhitley, Mrs. Joseph E.
Twin Castle Apts. ....Winston-Salem
Vhitt, Mrs. Walter Fuller, Jr.
206 Charleston ....Monroe
Vhittington, Mrs. Claude Thomas
600 Country Club Dr. ....Greensboro
Viedman, Mrs. James
Baldwin Woods ....Whiteville
Viggins, Mrs. John Carroll, Jr.
1949 Club Park Rd. ....Winston-Salem
Vilder, Mrs. Roboteau Terrell
Rotary Drive ....High Point
Vilkerson, Mrs. Charles Baynes, Sr.
517 N. Wilmington St. ....Raleigh
Vilkerson, Mrs. Charles Baynes, Jr.
2113 Woodland Ave. ....Raleigh
Vilkerson, Mrs. Jack Winfield ....Stantonsburg
Vilkerson, Mrs. Louis Reams
2301 Dixie Trail ....Raleigh
Vilkins, Mrs. Kenneth Worth
102 S. Pineview Ave. ....Goldsboro
Vilkins, Mrs. Robert Bruce
1007 Minerva Ave. ....Durham
Vilkinson, Mrs. Charles Tolbert
521 S. Main St. ....Wake Forest
Vilkinson, Mrs. James Spencer
3029 Granville Dr. ....Raleigh
Vilkinson, Mrs. Louis Lee
1033 Rockford Rd. ....High Point
Vilkinson, Mrs. Robert Watson, Jr.
513 S. Main St. ....Wake Forest
Vill, Mrs. Thomas Augustine
207 N. Hoffman St. ....Dallas
Villett, Mrs. Robert Walter
Galax Dr., Route 6 ....Raleigh
Villiams, Mrs. Charles David, Jr.
536 Seneca Place ....Charlotte
Villiams, Mrs. Charles Frederick
3203 White Oak Rd. ....Raleigh
Villiams, Mrs. David A.
Anson Avenue ....Laurinburg
Vililams, Mrs. Edward Jerome
301 Lancaster ....Monroe
Villiams, Mrs. Ernest Council
1008 Edgewood Circle ....Gastonia
Villiams, Mrs. Jerome Otis
105 Country Club Dr. ....Concord
Villiams, Mrs. Kenan Banks
747 Oaklawn Ave. ....Winston-Salem

Williams, Mrs. Leonidas Polk
300 S. Granville St. ....Edenton
Williams, Mrs. McChord
3954 Churchill Rd. ....Charlotte
Williams, Mrs. Ralph Bertram, Jr.
714 Forest Hills Dr. ....Wilmington
Williams, Mrs. Robert
2305 Hathaway Rd. ....Raleigh
Williams, Mrs. Robert Weser
727 Windsor Dr. ....Wilmington
Williams, Mrs. Roderick Thomas ....Farmville
Williams, Mrs. Samuel Clay
201 Westview Dr. ....Winston-Salem
Williams, Mrs. Samuel Hodges, Jr.
Bath Highway ....Washington
Williams, Mrs. Thomas Richard, Jr.
25-9th Ave. N.E. ....Hickory
Willis, Mrs. Candler Arthur
Route 2 ....Candler
Willis, Mrs. Harry Clay
906 W. Vance St. ....Wilson
Willis, Mrs. Tom Vann ....Sparta
Willis, Mrs. William Henry, Jr.
Riverview Park ....Bridgeton
Wilsey, Mrs. John Derrick, III
Reynolda Estates ....Winston-Salem
Wilson, Mrs. Clarence Lafayette
212 N. Main St. ....Lenoir
Wilson, Mrs. Frank
2317 Hathaway Rd. ....Raleigh
Wilson, Mrs. Franklin LeRoy
1908 Sharon Rd. ....Charlotte
Wilson, Mrs. Hadley McDee
117 Woodland Dr. ....Boone
Wilson, Mrs. James Stepheson
1501 Washington St. ....Durham
Wilson, Mrs. John Knox
1008 Dover Rd. ....Greensboro
Wilson, Mrs. Samuel Allen
710 E. Park Dr. ....Lincolnton
Wilson, Mrs. Thomas Barnette
3328 White Oak Rd. ....Raleigh
Wilson, Mrs. Walter Howard
2017 St. Mary's St. ....Raleigh
Wilson, Mr.s William L.
114 Byron Place ....Raleigh
Winkler, Mrs. Harry
1931 Ferncliff Rd. ....Charlotte
Winstead, Mrs. Ellis Grey, Sr. ....Belhaven
Winstead, Mrs. John Lindsay
302 Greene St. ....Greenville
Wise, Mrs. Fred Eugene, Jr.
1509 Maryland Ave. ....Charlotte
Witherington, Mrs. Dexter T.
Greenbriar Rd. ....Kinston
Withers, Mrs. Syndor Terry
701 W. Vernon Ave. ....Kinston
Withers, Mrs. William Alphonso
2411 Country Club Dr. ....Raleigh
Witten, Mrs. Ernest Robert Sidney
80 Wembly Rd. ....Asheville
Wolfe, Mrs. Harold Eugene
300 S. Andrews Ave. ....Goldsboro

Wolfe, Mrs. Hugh Claibourne
108 Beverly Place .... Greensboro
Wolfe, Mrs. Nathan Carl, Sr. .... Burgaw
Wolfe, Mrs. Ralph Verlon
440 N. Hawthorne Rd. .... Winston-Salem
Wolff, Mrs. Dennis Roscoe
Cannon Court Apts. .... Greensboro
Wolff, Mrs. George Thomas
805 Magnolia St. .... Greensboro
Womble, Mrs. Edwin Cornelius .... Wagram
Womble, Mrs. William H., Jr.
Westridge Rd., Route 9 .... Greensboro
Wood, Mrs. Ernest Harvey, Jr.
Fern Lane .... Chapel Hill
Wood, Mrs. Frank
115 W. Church St. .... Edenton
Wood, Mrs. George Thomas, Jr.
Route 1 .... High Point
Wood, Mrs. Hagan Emmett
W. N. C. Sanatorium .... Black Mountain
Wood, Mrs. Sherrod Newberry .... Enfield
Wood, Mrs. William Lupton, Sr.
Box 278 .... Yadkinville
Wood, Mrs. William Reed
204 Rockford Rd. .... Greensboro
Woodard, Mrs. Marshall Wayne
145 Midland Dr. .... Asheville
Woodburn, Mrs. Clark Harold .... Littleton
Woodhall, Mrs. Maurice Barnes
4006 Dover Rd., Hope Valley .... Durham
Woodruff, Mrs. Paden Eskew
1732 Brenner Ave. .... Salisbury
Wooten, Mrs. Cecil William, Jr.
1101 Rhem St. .... Kinston
Wooten, Mrs. Floyd Pugh
1114 W. College St. .... Kinston
Wooten, Mrs. John Lemuel
Circle Drive .... Greenville
Wooten, Mrs. William Isler
Maple St. .... Greenville
Worde, Mrs. Boyd T.
504 Carver St. .... Durham
Worden, Mrs. Neil Ashton .... Hope Mills
Worth, Mrs. Thomas Clarkson
500 Lake Boone Trail .... Raleigh
Wrenn, Mrs. Richard Nickles
1432 Ferncliff Rd. .... Charlotte
Wright, Mrs. Charles Newbold .... Jarvisburg
Wright, Mrs. Frederick Starr
933 Hendersonville Rd. .... Asheville
Wright, Mrs. Isaac Clark
329 Transylvania Ave. .... Raleigh
Wright, Mrs. James Rhodes
3319 White Oak Rd. .... Raleigh
Wright, Mrs. James Thurman .... Belhaven
Wright, Mrs. Samuel Martin
2003 Morganton Rd. .... Fayetteville
Wright, Mrs. Thomas Hasel, Jr.
555 Hempstead Pl. .... Charlotte
Wright, Mrs. William David
1222 Grayland St. .... Greensboro
Wyche, Mrs. Joseph Thomas
Baldwin Woods .... Whiteville
Wylie, Mrs. William DeKalb
310 Arbor Rd. .... Winston-Salem
Wyngarden, Mrs. James B.
1104 Knox St. .... Durham

Yeomans, Mrs. Merrill Brooks
403 Gold St. .... Shelby
York, Mrs. Shelley C., Jr.
400 Randolph St. .... Thomasville
Young, Mrs. Charles Gibson
306 Kirk Road .... Greensboro
Young, Mrs. David Alexander
1546 Iredell Dr. .... Raleigh
Young, Mrs. John Paul
18 Colonial Place .... Asheville
Young, Mrs. Joseph Alexander
342 S. College Ave. .... Newton
Young, Mrs. William Beauregard
Woodland Drive .... Wilson
Young, Mrs. William Glenn
1407 Dollar Ave. .... Durham
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The Wilburt C. Davison Number

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NORTH CAROLINA MEDICAL JOURNAL

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Dedication

The Medical Society of the State of North Carolina takes great pride in honoring one of its most distinguished and beloved members by dedicating this October issue of the NORTH CAROLINA MEDICAL JOURNAL to Dr. Wilburt C. Davison. All papers published in this issue have been contributed by former students and staff members on the Pediatric Service at Duke University Medical School during the time that Dr. Davison was professor of pediatrics and chairman of the department. Limitations on the number of publications have been necessary because of the lack of space, and it is with regret that more of the papers contributed could not be published.

The Editors of the JOURNAL join all in this salute to Dave.

W. M. NICHOLSON, M.D.
Duke University Medical Center

The Davison Saga

In January of 1927 Dr. Wilburt C. Davison was chosen to plan, organize, build, and direct the new Duke University School of Medicine and Medical Center. In the subsequent 33 years, he has guided this institution to an enviable position in the roster of the world's greatest medical establishments. As Dean, James B. Duke Professor of Pediatrics, and Chairman of the Duke University Medical Center, Dr. Davison has left the indelible imprint of his vision, vigor, and versatility upon his university, his state, and upon medical education and medical care everywhere.

This great accomplishment is but part of the Davison saga. Sensitive to the public and hospital problem of financing hospital care, Dr. Davison was a proponent of methods of prepayment as early as 1928. The fruition of his concept of a community-wide program, however, was delayed by the stock market crash of 1929. Had this early effort been successful, the Durham Hospital Association would have been the first prepaid voluntary hospital service plan in the United States. Nevertheless, as a result of his efforts, two of the earliest Blue Cross plans in the nation were established in North Carolina: one in 1932 and one in 1935. Moreover, his pioneer work contributed significantly to the development of the National Blue Cross program, which today covers more than 56,000,000 people and which paid hospitals and doctors approximately $2,000,000,000 in 1959. The tremendous success of the Blue Cross program is a fitting tribute to this dynamic man whose vision and courage helped to make it possible.

As a result of his extensive administrative experience, Dr. Davison concluded that there was a great need for more efficient management in hospitals. When the Duke Medical Center was opened in 1933, he inaugurated a formal program for the training of hospital administrators. In this program he anticipated the community hospital development program being launched by the Duke Endowment in the Carolinas and by the Hill-Burton program throughout the nation. Both programs required trained administrators with a comprehensive understanding of hospital operation and a thorough orientation in the health fields.

Throughout a distinguished career as physician, educator, author, administrator, Dr. Davison has consistently maintained that, while specialization in medicine is necessary, the success of total medical practice must depend upon a substantial portion of well rounded generalists. Because of his numerous contributions to the development of general practice, he was awarded the Certificate for Meritorious Service Award of the American Academy of General Practice in 1960.

After 34 years Dr. Davison has stepped down as the first and only Dean of the Duke Medical School. Now he has relinquished his duties as former chairman and professor of pediatrics. A great era has come to an end; yet the Davison imprint is permanently set. His contribution to Duke, to his profession, and to the advancement of medicine is incalculable. While many helped at the onset and during the intervening years, the present Duke Medical Center, in all that it has accomplished and in its still expanding nature, stands as its own tribute to the man who planned, organized, and directed the Medical School from the ground up and guided it to the place of prominence which it now holds. During Dr. Davison's tenure the Medical Center graduated some 1,800 doctors, engaged in endless research, treated and saved the lives of thousands of patients, saw the medical profession rise to new and higher standards, and developed its own new dean to take Dr. Davison's place and to carry on with his vision, philosophy, and sense of responsibility.

An engaging, dynamic individual, admirable in every way; a scholar, yet a practical clinician and an administrator with a great heart, a great mind, and a great personality—Dr. Davison eminently deserves the many honors that have been accorded him over the years.

Jay M. Arena, M.D.

Wilburt C. Davison, M. D.

A Few Reminiscences About Dean Davison

GEORGE R. ANDREWS, M.D.
WAUSAU, WISCONSIN

One of the most memorable features of a medical education at Duke from 1930 to 1960 was Dean Wilburt C. Davison. Though it was of necessity rare for organisms orbiting around the transverse colon and the island of Reil to encounter someone who went as casually to Bangkok, Vladivostok, and Sydney as most people did to the A. & P., still Duke students always knew the Dean was there with them and for them.

His secretary might explain that at the moment he was in Washington or Peking, but such reports were never unsettling, however seemingly urgent the errand on which one had come. The image of the Dean winging sedately across the Steppes of Asia, over the oceans and continents, was somehow reassuring. It gave one the feeling that not only the concerns of American medicine, but somehow the ultimate interests of Duke itself stretched to the furthest corners of the planet and were in the best possible hands. While students and faculty were straining together to push back the frontiers, the Dean was out surveying new ones against which future generations could organize alarums and expeditions. He provided a feeling that we were participating with him as he presided over the course of empire and the unfolding of manifest destiny on the most elevated level of medical affairs.

The reason for this sense of close identification with him was the air of warm and hearty interest he always showed to each student who came to see him. It was as true in the years after graduation as before. If you came to lament a low-pass, the Dean was as dismayed as you were at such an obvious miscarriage of justice. If you came to ponder the temptation of internships and residencies which tendered the lures of living wages, he was gently sympathetic but nonetheless judicious in pointing out the large price of a lower quality of training which would most likely have to be paid eventually. For the alumnus returning to Duke, the feeling on seeing Dean Davison was that of meeting an old friend rather than someone who had once intimidated on the basis of a superior office and the power to control the destiny of the students within his sway.

Someone once defined medical meetings as places where doctors are given the opportunity to act for a few days like the men they once imagined they would be. There are times when, to most students, medical schools seem like places where some physicians are permanently provided such an opportunity. Of course, such cynical critiques need to be softened by acknowledgment of the somewhat paranoid state of mind of the average medical student, who is momentarily expecting disaster, trembling at the shadows of leaves falling past the window in October, and hearing whispers in the night as portents of calamity. In the depths of their being, few of them ever really expect to graduate or become licensed physicians.

In such an atmosphere of highly charged suspense and imminent catastrophe, the sight of Dean Davison was more salutary than two or three years on an analyst's couch would have been. With collar open and loosened tie, he was the picture of ease and common sense. He was an invitation to learning. The omnipresent pipe completed, though nothing could have improved, the picture.

Equally as effective as the sense of existing in a meaningful relationship to the world and its affairs which Dean Davison provided was a historical dimension he gave in which, through a sort of benign "guilt by association," we all could participate.

As a Rhodes scholar he had had first-hand experience of England and its institutions of higher education in a period preceding the first World War. His professional association and friendship with Sir William Osler had a certain magical quality about it. The thought that the Dean had been so closely associated with this legendary figure in medicine was redemptive for young men and women on whose horizons usually loomed only such grave concerns as learning the names of all the femur's tuberosities, or looking up and memorizing every article on ainhum ever written.

The uncounted pictures of athletic teams, military figures, educators, and distinguished colleagues of the past, which took the place of wallpaper in the Dean's office, gave a visitor the feeling of being immersed in the historical process. The fact that he had been a member of the faculty of Johns Hopkins and had led the Duke faculty in creating the Duke Medical School gave a final measure of distinction.

The peripatetic nature of the Dean's Office through the years at times seemed to support rumors periodically circulated that he was voluntarily withdrawing from his headquarters to "make room for mice and seagulls" in the interest of research. The sight of the Dean and his faithful associate, Carl, heading undaunted out to the periphery of the campus in search of a new, more secure base of operations was a familiar and moving one for students through the decades. When at last the final location was once more near the lobby of the medical school, even though on the opposite side of the hall from where it had been for such a long time. there was a sense of fulfillment. There was a rightness about the return which had all the components of the "happy ending." The Dean had come home again where he belonged. The dramatic unities had been fittingly observed.

The Dean's Portrait

Probably every Duke Medical School graduate has some particular memory of the Dean which he particularly cherishes. If I had to choose one, it would be of a singular experience, which several of my classmates and I shared with him. One summer's day I was walking innocently toward Howland Ward when I encountered Dr. Grant Taylor, who was the Assistant Dean at the time. He reached out and beckoned. I paused uneasily, expecting some clap of doom. such as that in 10 minutes I was to present a case in the amphitheatre to the student body, faculty, and visiting luminaries from Harvard, Hopkins, and Aberdeen.

"Get a few of the boys," he said. "They're painting a picture of the Dean, and they need some of you fellows to sit around at his feet like on rounds."

It was at the moment a seemingly obscure assignment, since none of us had ever had any experience along these lines, but it sounded like a splendid opportunity to break the routine. I had no idea how long it took to paint a dean's portrait, but it sounded as though we might get a few days of official leave while interns and residents fumed impotently at our departure from the chores of the lab and the ward. At the particular moment it had not clearly dawned on me what a remarkable enterprise we were embarking upon, nor what a privilege we were being offered.

I mentioned the project to Hank Gowdy, Bill Hadley, and Charles Gunn, who responded with enthusiasm. We drove over to the building on the East campus where we were to meet. We were in the kind of holiday mood students always develop when unexpectedly school lets out, whether because of plagues, wars, natural catastrophes, or some equally providential but much more pleasant reason such as the mission on which we engaged.

It was warm weather, clear and sunny. We were all wearing our white coats, so that we would look like, even if we were not altogether so, authentic students in the painting. The room was a large one on the ground floor, the setting simple and, as I look back on it, similar to the kind of scene we have all observed countless times on our TV screens.

There was a tall wooden stool for the Dean. In front of it were four folding chairs without backs for us. The total effect was quite reminiscent of rounds with Dean Davison on the pediatric ward. The easel was set up at a distance of perhaps 30 feet from the Dean. He faced us and the artist, and we faced the Dean. It gave the artist a fine view of him and of our backs, though there was a third or quarter view of the faces of the students on the ends, as the chairs were arranged in a curving line for purposes of composition.

Dean Davison was there talking to the painter. We were introduced to him, and then all of us, feeling remarkably self-conscious, sat down. In a little while we had grown accustomed to the situation, and there began the most remarkable week any of us had known in medical school. Of course, there was no work involved except sitting for a couple of hours, morning and afternoon, on those backless chairs. During the entire time, the Dean reminisced about events in his life with the relaxed, keen sense of humor which is one of his most notable characteristics. He provided us with glimpses of existence at Princeton, in England when he was a Rhodes Scholar, at Hopkins and innumerable places around the world where he had been in his work and travels. He sketched personalities, both distinguished and uncelebrated, that he had dealt with through the years. He spoke of medical schools, his philosophy of education, his views on the microcosm and the macrocosm. There were the highly amusing anecdotes which he always has in such limitless supply.

One of the many that I recall was of a trip he and Dr. Perlzweig, the first chairman of the Department of Biochemistry, made to Russia in the early thirties. Their hosts suggested at one point that they all take a day off at the beach. The two travelers said that unfortunately they hadn't brought bathing suits. This didn't matter, their hosts told them, since it was not the custom to wear anything on the beach in that part of Russia. Somewhat nonplussed, but not wishing to seem unappreciative of the offer, they accepted and went along. As Dean Davison recalled it, he and Dr. Perlzweig both spent a rather amusing day covertly watching each other in the process of trying to act casual and relaxed, though under circumstances which, in at least one respect, were strikingly unfamiliar to them.

"Actually," the Dean told us, "I didn't regret the occasion myself because I had always been curious about how the Russian women were built." He paused and puffed meditatively on his pipe while we waited. He looked at us then with a twinkle in his eye. "Very solidly," he concluded. "Very solidly."

By chance I sat second from the left, with Gowdy on my left, Bill Hadley on my right, and Gunn on Hadley's right. Bob Sheridan, as I recall, replaced Charles after the first day or two because he couldn't return. As it turned out, the man on the extreme right wasn't included in the painting, and only a portion of Gowdy's and Bill's faces showed up. Mine didn't at all, since I had my back to the painter.

The week passed very swiftly, and we were all soon back at our familiar labors. The painting was finished in detail without further help from us. As everyone knows, it now hangs in the amphitheatre at Duke, where countless generations of students have a splendid opportunity to see what the school's first Dean looked like in those days when he ran the institution, traversed the globe, and shepherded the successive bands of aspiring physicians through the innumerable psychic and physical traumata of undergraduate days.

It is just the right sort of portrait to have painted. It catches and preserves faithfully the personality of the Dean, with his warmth, his dislike of pomp and formality. The loosened tie, the open collar, the pipe symbolize perfectly the kind of man and teacher he was and is. Someone once described the ideal school as a log with Mark Hopkins on one end of it and the student on the other. In more modern terms, an ideal medical school might be defined as a half dozen stools with Dean Davison on the highest and the students on the others.

Outside of the fond recollections of that week with the Dean while we were all being painted, the anonymous students who posed with him have had a unique and very interesting little psychologic problem. Whenever they drop by and see the painting, as well as at other times, they realize that it is their singular role to advance backwards upon posterity through the eons, sitting forever at the feet of Dean Davison. While this might have caused some slight emotional difficulty in getting up on their feet and facing the world, the problem has not been overwhelming. But however great the encouragement to remain dependent throughout eternity, or at least until the amphitheatre collapses, the handicap has been far more than compensated by the uncancellable opportunity of dropping in and seeing captured forever a brief moment out of youth, spent in the company of a singularly knowledgeable and inspiring man whose abilities as a teacher were surpassed only by his capacities as a human being.

Wilburt C. Davison -- The Dean and the Friend

An Appreciation by a Latin American Pediatrician

Ernesto Cofino, M. D.
Guetemala, Central America

It would be a big job to relate, even briefly, the scientific trajectory and the magnitude of the work of Dr. Davison, even when we follow him only as a pediatrician, the founder and dean of a medical school, and the author of one of the most original summaries of pediatrics ever written. I am sure that a more complete picture will be developed in this special dedicatory issue, by his intimate colleagues, by his pupils from yesterday and today, and by those who have lived close to him and know of his life of continual movement, his immense spirit of understanding, and his dominant obsession of helping others even before their petitions.

My contribution to this "honor" number will be simple, considering only Dr. Davison's position in international pediatrics, especially in Latin America.

First of all, I want to say that one of the things that have characterized him is his fineness. The generous help he gave was always invisible, so that the people who received it seemed to walk by their own efforts.

Dr. Davison has passionately loved to travel—a characteristic of his constant activity and his interest in all countries and people. He has gone everywhere with his eyes open and with the fervent wish to understand and appreciate all the good, noble, and interesting things that every country offers.

He has been able to accomplish much by a kind of intuition that is an expression of his genuine generosity. To understand the dominant feelings of a country, to capture its virtues, and to excuse its defects is very difficult when the language of the country is well known: but to do this without knowing the language is possible only to men provided with a sixth sense—the sense of love for all the inhabitants of this planet. I really think that if Dr. Davison had the opportunity to travel to other planets, he would know how to interest and understand the inhabitants and to serve them.

How I Met Dr. Davison

In 1944 I accepted the generous invitation of the State Department of the United States to visit representative pediatric clinics in my position as professor of pediatrics in the University of Guatemala. I was anxious to meet personally the North American pediatricians and professors, having followed with interest the notable development of pediatrics in that continent through books, publications, and occasional assemblies. My own training in medicine and pediatrics I owed to France, where I studied in the school of Professor Robert Debré.

I arrived in Washington in June, 1944, and started getting acquainted through the courtesies of Mrs. Elizabeth Shirley Enochs and Professor Henry Helmholz (who died recently). It was my first formal visit to the United States, and I was a little confused, mainly because of my incomplete knowledge of the language. Professor Helmholz discussed several possibilities for permitting me to gain a better understanding of North

American pediatrics, allowing me to choose the most convenient method of meeting my needs. We came to the conclusion that the best place to begin was at Duke, under the direction of Professor Wilburt C. Davison.

So I came to Durham at the beginning of July, 1944, finding myself in a very small town, with a university which had been set up by the generosity of one man—an extraordinary university town, in which the contributions of Dr. Davison had been remarkable.

Next day I was in the presence of the Dean in an environment of frankness and simplicity that contrasted with the solemnity and grandeur of the severe English-style buildings. I was introduced immediately to the Dean's office; it was a simple room full of bookcases, pictures, caricatures, and diplomas. Everything was a palpable and graphic representation of Dr. Davison's life, his love of travel, his passion for people, and his vocation.

The Dean's personality was in harmony with his environment: on a revolving chair, behind a simple but ample desk, I saw a man big in his physical dimensions, the only ones I could see then. The same dimensions I afterward saw as small in comparison with his spiritual ones. He received me as he used to when he did not have to submit to rules and protocol—wearing a shirt without a tie, and with a pipe in his mouth. That famous pipe was my nightmare for several months; because of it his words came out of a small space, causing me to exert constant effort in order to understand a little of his speech.

Going to an unknown country, where the culture, customs and way of life are different from one's own, is always most difficult, especially when one does not travel as a tourist with the pockets full and the mind empty. When the wish is to learn and retain what a generous country has to offer, the differences in language and customs are great obstacles. This situation is even harder when one is past his youth and has enjoyed in his native country a satisfactory social, medical, and educational position. It is difficult to become a student again, and terrible in the American sense. For Americans, in order to save time, use a great number of abbreviations, well known to them but absolutely incomprehensible to the poor beginner.

So it is not surprising that my face took on the proper characteristics of an idiot, and that the resident or "boarder boy," not used to dealing with foreign doctors and apparently confusing language difficulties with ignorance, asked, "Who is this Guatemalan professor who does not begin to understand pediatrics?" For the foreign doctor the fight was very hard, because he did not want to develop an inferiority complex.

Great was the confusion and the mental exertion of the "poor foreign professor" after he had been on visits, in discussions, conferences, and conversations in the dining room.

But then Dr. Davison's intervention began to be felt. With the goodness of a friend, the knowledge of an expert, and all the weight of a dean, he smoothed out all the harshness and unpleasantness, and did it all without the visitor's even realizing that someone was trying to help him.

The Magic Touch

Dr. Davison intervenes in everything, but the foreign visitor does not feel his presence. He has the impression that, thanks to his own spirit, his difficulties have become less and his initial inferiority complex is replace by a feeling almost of superiority. He feels that his Latin spirit has given him an extraordinary capacity for adaptation. He feels that doors are opened by a magic eye as soon as he steps near. In every department and office there is someone ready to help, almost guessing his questions. He doesn't realize that this great man, Dr. Davison, who has the virtue of "resting—working," this man who knows how to fill his "leisure" time, is behind all this attention.

This attention is not limited to the medical and technical aspects of the visitor's life, but is in the social atmosphere too, letting him know the way of life in North American homes. To attain these results, Dr. Davison starts inviting the visitor to his house and honoring him with special attention. His colleagues, perfectly trained in his school, follow the Dean who has exhibited such a comprehensive and helpful attitude.

One knows that Dr. Davison wants his department always to be in good harmony. Besides, everybody knows that in him we have the teacher and the friend to solve all our family, economic, and educational difficulties. From him we have learned to follow the precept, "Don't let your left hand know what your right hand does."

It was under these conditions that I stayed at Duke for four months, being forced to leave to complete my program of study.

When I went to Mayo Clinic with Dr. Helmholz, to the University of Minneapolis with Dr. McQuarrie, and afterwards to Chicago, New York, and Richmond, my situation had changed. I was more sure of myself because I had a greater knowledge of the language and had become acquainted with North American medical concepts; but most of all, I had captured the peculiar way that North Americans have of helping others: "Few words and much efficiency."

"A Special Place"

After this trip I had many occasions to meet Dr. Davison—in assemblies, meetings, when he visited my country, and in his home at Duke. Every time I have been able to appreciate his extraordinary qualities. Among all the good people I have been privileged to know, he has a special place. I am sure that this feeling is unanimous among his many friends and admirers in Latin America.

The man who was able to create a medical school in a tobacco field; the man who wrote the most original and useful book on pediatrics that exists; the man who has visited so many places and appreciated all the good in them; the man who knows how to create, just by his presence, an atmosphere of cordiality and human understanding; a man with this moral in this materialistic world—Dr. Davison keeps playing a role of top quality even when regulations impel him to retire from some of his university activities.

His years have been full, and his crop very good. Now that he leaves the routine administrative responsibilities, he will be able to practice even more freely his role of furthering good will among peoples.

. . . after unmeasured centuries of vague and meddlesome interference, restless speculation and error sustained by convinced authority, Hippocratic medicine begins to introduce studious habits into the flimsy intellectual life of mankind. And at the same time a code of honor, a creed for internal self-government was established by a group of men devoting themselves to helping their fellows. We can be proud that physicians, even as they were making a start toward science, became the first and remain the only profession with a self-imposed creed for monitoring their own acts. Its failures remind us that physicians are human. not supermen.—Bean. W. B.: Some Historic Sidelights on Medicine's Forgotten Man—The Patient. The Association of Life Insurance Medical Directors of America, 1961.

Theoretical and Practical Implications of the Epidemiologic Differences Between Acute Rheumatic Fever and Acute Nephritis

Lewis W. Wannamaker, M.D.*
Minneapolis, Minnesota

Clinically, acute rheumatic fever and acute nephritis bear little resemblance to each other. In the former, the presenting symptoms may be migrating joint pains, carditis, choreiform movements, or characteristic skin lesions. In the latter, the chief manifestations may be bloody urine, edema, hypertension, convulsions, or heart failure. Yet both diseases arise from a common type of infection.

Evidence linking acute rheumatic fever and acute glomerulonephritis with streptococcal infections has become steadily more convincing during the past several decades. These organisms can be incriminated on clinical, bacteriologic, serologic and epidemiologic grounds[1]. Consequently one can now state with more certainty than is ordinarily prudent in clinical medicine that any patient with either of these two diseases has been recently infected with a group A beta-hemolytic streptococcus. Sometimes the patient will have had a previous exudative tonsillitis or scarlet fever. In such instances the clinical sequence of infection followed by rheumatic or nephritic complications is striking and obvious. As often as not, however, the preceding infection is asymptomatic, mild, or atypical, so that without laboratory studies or a history of a typical clinical picture in another member of the family, the streptococcal illness goes unrecognized. Even when clinical evidence of streptococcal infection is lacking, these patients almost invariably show antibody titers which indicate a recent streptococcal infection[2]. In unusual instances, patients may have gross hematuria and clinical evidence of a mild acute nephritis which is apparently unrelated to streptococcal infection[3], but certainly the great majority of cases of acute glomerulonephritis and probably all cases of rheumatic fever are so related[1, 4].

With the increasing awareness of the regularity with which acute streptococcal infections precede the development of acute rheumatic fever and acute nephritis, the question has naturally arisen as to why some patients come down with these complications while others escape them. Equally puzzling is why some patients have one complication whereas others have another. If both diseases are initiated by streptococcal infections, it is also strange that they are so rarely seen in the same patient.

The role of infection and the similarities and dissimilarities of these two diseases make a fascinating detective story. Although the story is not yet complete, the unraveling to date has important implications concerning the diagnosis, manage-

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From the Department of Pediatrics, University of Minnesota, Minneapolis 14, Minnesota.

*Career Investigator of the American Heart Association and Professor of Pediatrics, University of Minnesota School of Medicine.

This interpretative review has been written to honor Dr. W. C. Davison, whose continued efforts to bring young men from small towns in the Southeast face to face with the problems of pediatrics and medicine have reaped benefits which are widely distributed geographically and quite varied in their expression. It was as a student and house officer on the pediatric wards of Duke Hospital that the author's attention was first drawn to the frequency of skin infections in acute nephritis, and it was during his rotation as a pediatric intern in the bacteriology laboratory that he first struggled with skin cultures from these patients in an attempt to isolate beta-hemolytic streptococci from staphylococci, which even in those days were already beginning to show a tendency to take over. In this small way, the author would like to acknowledge the importance of those formative years and of the medical teachers and educators at Duke, particularly Dean Davison, who guided him through them.

The work from the author's laboratory referred to in this paper was conducted, in part, under the sponsorship of the Commission on Streptococcal Diseases, Armed Forces Epidemiological Board, and was supported by the Offices of the Surgeons General, Department of the Army, Washington, D. C., by a research grant (H-1829 C4) from the National Heart Institute of the National Institutes of Health, U. S. Public Health Service, and by the Minnesota Heart Association.

ment, prognosis, and prevention of these diseases.

Epidemiology

Regarding the epidemiology of the two diseases, table 1 shows that they have many features in common. Both are preceded by a streptococcal infection. In both, the infection is followed by a latent period before symptoms of acute disease become manifest. Both diseases tend to occur in families. Both are more common in children of school age, and both flourish under crowded living conditions in low economic areas. They are frequently seasonal in incidence, the majority of cases appearing in the late winter or spring. They both may occur in epidemics, causing great concern in schools and military installations.

Table 1
Common Features of Acute Rheumatic Fever And Acute Nephritis

Preceded by streptococcal infection
Latent period
Tend to occur in families
More common in children
More prevalent under crowded, low economic living conditions
Frequently seasonal
May occur in epidemics

Many of the common features of these two diseases can be explained on the basis of the first. For example, acute rheumatic fever and acute glomerulonephritis are more common in children because streptococcal infections occur more often in this age group. The peak incidence of the two diseases—at about 6 or 7 years of age—is almost identical with that of scarlet fever and other streptococcal infections. The crowded living conditions encountered in low economic areas are known to be conducive to the spread of streptococcal infections[5]. Their seasonal incidence is readily explained by the variations in seasonal occurrence of streptococcal infections. Moreover, their tendency to appear sometimes in epidemics has been directly related to the epidemic occurrence of streptococcal infections. Some of these features, such as the latent period, familial occurrence, and age distribution will be referred to again; for, although superficially similar, they show certain differences which may be important.

Differences

Table 2 shows some of the differences in the epidemiologic features of these two diseases. In acute rheumatic fever, the site of the preceding infection is almost always in the pharynx or tonsils. In acute nephritis, although the preceding infection may arise in the throat, it is frequently located on the skin. For example, in a study of 73 patients with acute glomerulonephritis admitted to the pediatric service of Duke Hospital, approximately half had a preceding pyogenic infection of the skin without evidence of a preceding respiratory tract infection[6]. In rheumatic fever the initiating infection is almost always superficial. Many clinicians have observed how rarely rheumatic fever follows deep suppurative infections as compared with the relative frequency of acute nephritis[7].

Although both diseases have a latent period, its average duration is 18 days in acute rheumatic fever, but only 10 days in acute nephritis[8].

The attack rates for the two diseases differ. The yearly incidence of acute rheumatic fever varies less than that of acute nephritis, being more closely related to that of scarlet fever[7]. On the other hand, the incidence of acute nephritis may fluctuate strikingly. During World War I "trench nephritis" became epidemic in Europe, with more than 1,000 cases reported in the British Army alone[9]. During World War II little or no nephritis was observed in the military forces, although rheumatic fever was prominent[10].

Following known streptococcal infections the attack rate for acute nephritis is quite variable, ranging from zero to 18 per cent[11]. In contrast, the attack rate for rheumatic fever following strictly defined streptococcal infections is quite constant—at about 3 per cent. This rate does not vary with the geographic area, the time of infection in relation to the stage of the epidemic, the nature of the clinical manifestations of infection, or the serologic type of the group A streptococcus[12].

Table 2

Differences In Acute Rheumatic Fever And Acute Nephritis

Features	Rheumatic Fever	Acute Nephritis
Infection		
Site	Throat	Often skin
Type	Superficial	May be deep
Latent period	18 days	10 days
Attack rate	Constant	Variable
Recurrences	Common	Rare
Familial occurrences	Staggered	Simultaneous
Age (infants)	Rare	Not infrequent
Geographic distribution	North	North and South

Recurrences of rheumatic fever are common. They are a dramatic and important part of the natural history of the disease. By contrast, once a patient recovers from acute hemorrhagic nephritis he rarely has another attack[13]. Some physicians have suggested that such individuals are less susceptible to nephritis than is the general population[8].

Differences in the familial incidence of the two diseases is interesting. Acute nephritis tends to occur in outbreaks, so that several cases may appear in a family within a short time. Rheumatic fever rarely appears simultaneously in two or more members of the same family, although it not infrequently does so at different times or in different generations[14].

The geographic distribution of these diseases is different[7]. Clinical rheumatic fever appears to be more common in the Northern than in the Southern states, but acute nephritis seems to be equally distributed.

Although the age distribution of the two diseases is in most respects quite similar, there seems to be a difference in the lowest age groups. Acute rheumatic fever is extremely rare in young infants, whereas acute nephritis, although not a common disease of infants, is relatively frequent under the age of 2. For example, in a study of 400 cases of acute nephritis, one author has reported 47 cases in children under this age[15].

Etiology

There has been much speculation regarding the significance of these obvious differences. Both host and bacterial factors have been considered as possible etiologic explanations.

Host factors

The failure of these complications to develop in many patients with streptococcal infections suggests the importance of host factors. The tendency for rheumatic fever to occur in several generations of the same family suggests a genetic component[14]. As yet no specific genetic defect has been identified, and as in tuberculosis it is difficult to separate inherited from acquired host differences. There appears to be no doubt that patients with rheumatic fever react differently to streptococcal infections than does the normal population. The attack rate of rheumatic fever following well documented streptococcal infections is many times higher in individuals who have had a previous attack than it is in the normal population (up to 87 per cent[16] as compared with 3 per cent). Whether the first attack selects those of innately high susceptibility, or whether it is the first overt expression of an acquired modification of host response is not known. A clear distinction between these two possibilities will probably not be made until the specific nature of this difference has been identified and can be looked for in pre-rheumatic fever subjects and in rheumatic families.

Another difference between rheumatic and normal individuals that may be host-related is the increased antibody response of rheumatic subjects following streptococcal infections[2a]. The average increase in antibody titer is appreciably higher in patients who have rheumatic fever following streptococcal infection than in those who do not. However, some patients with rheumatic fever fail to show an antibody response to a particular antigen, and it is only by testing for several different streptococcal antibodies that evidence of a preceding infection can be obtained[2b].

Also, the reported differences in antibody responses following streptococcal infection in rheumatic subjects may be due in part to the difficulty in differentiating clearly between streptococcal infection and streptococcal carrier states, unless antibody response is used as a criterion of infection. Since antibody response is the factor being evaluated in these studies, it cannot be used to define streptococcal infections. In the absence of this criterion, the population group which escapes rheumatic fever may include some individuals who are merely chronic carriers, with superimposed non-streptococcal infection, whereas it is unlikely that the group which had rheumatic fever includes any who have not been recently infected with streptococci. Thus, if rheumatic fever patients do indeed show an exaggerated antibody response, it appears to be inconstant. Moreover, it is definitely limited to streptococcal antigens, since these patients show no increased response to such antigenic substances as diphtheria toxoid, influenza vaccine, and cholera vaccine[17].

Although superficially rheumatic fever and acute nephritis may appear to be allergic in nature, no definite basic link to those diseases of accepted allergic etiology has been established[4], and no increase in incidence of allergic diseases has been demonstrated among patients with acute rheumatic fever or acute nephritis. The rarity of acute rheumatic fever in infancy suggests that, in contrast to acute nephritis, sensitization by repeated exposure to streptococcal infection may be necessary for its development[18]. It should also be pointed out, however, that streptococcal infections occur less frequently and the diagnosis of rheumatic fever may be more difficult in this young age group.

Among the more popular current theories involving host response one can mention hypersensitivity[19], auto-immunity[20], persistence of the living streptococcus in a native or altered state[21], and persistence of specific streptococcal products which may be inefficiently handled or toxic[19b 22]. Although some evidence has been adduced to support each of these factors as possible contributors to the pathogenesis of rheumatic fever or acute nephritis, no direct proof has yet been obtained in human hosts. Moreover, no altogether satisfactory models of these diseases have been produced in experimental animals.

Bacterial factors

That bacterial as well as host factors are important in the etiology of these diseases now seems indisputable, although it is not known which of the large number of streptococcal products already described[23], or which of the perhaps even larger number as yet not identified, may contribute to the pathogenesis of these diseases. Figure 1 presents diagrammatically some of the known morphologic features, cellular components, and extracellular products of the streptococci and how they relate to the classification of these bacteria. In man, most streptococcal infections of the throat (and probably also of the skin) are due to hemolytic organisms in the group A category, and it appears that this is the only group which can produce infections leading to acute rheumatic fever or acute nephritis. Even in such a simplified diagrammatic form, this organism appears to be extremely complex, producing a variety of substances, some of which are enzymatic, most of which are antigenic, and many of which have specific biologic activities.

The surprisingly uniform attack rate of rheumatic fever following infection with different strains or types of streptococci[12] suggests that all or most group A strains can produce rheumatic fever. Therefore, the "rheumatogenic substance" must be common to all or most of these strains. Among the various specific substances produced by group A strains which have been suspected by different investigators are strep-

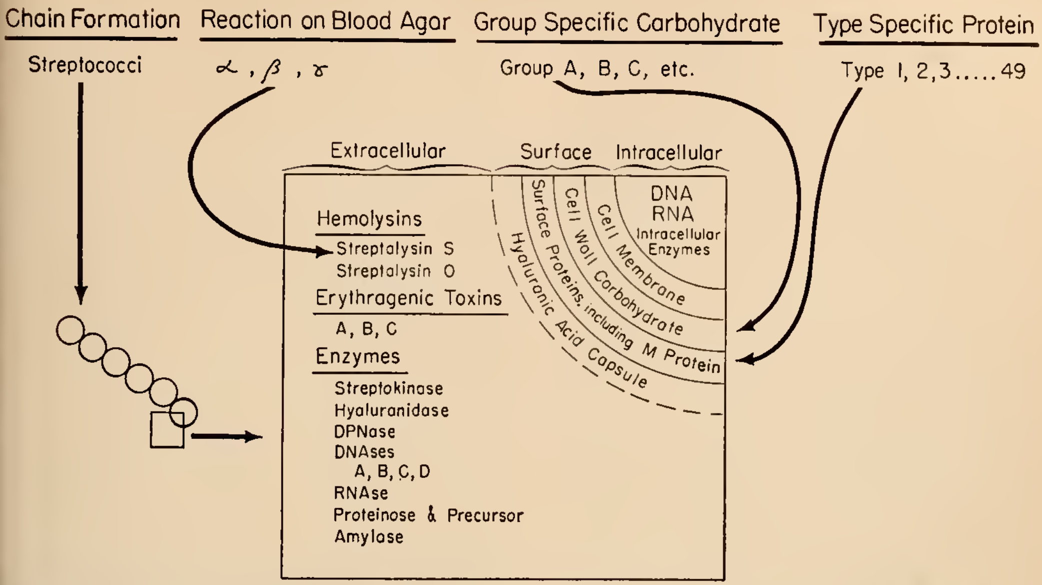

Fig. 1. A diagrammatic presentation of some of the known morphologic features, cellular components, and extracellular products of the streptococci in relation to the classification of these organisms.

tolysin S[4], streptolysin O[24], and the cell wall or group-specific carbohydrate[19b, 22]. The two hemolysins are produced by most strains of streptococci, whereas the latter substance is by definition the essential component of all group A streptococci. It is of interest that only one of these (streptolysin O) appears to be a true antigen in man, whereas the other two appear to be nonantigenic or poorly antigenic. Convincing evidence that these or any of the other common products of groups A streptococci are specifically concerned in the etiology of acute rheumatic fever has not yet been obtained.

Significance of Serologic Types

The important recent observations of Rammelkamp and colleagues[11 25] have elucidated some of the differences in the epidemiologic behavior of acute rheumatic fever and acute nephritis and have suggested the special significance of bacterial factors in the etiology of the latter disease. These workers have shown that acute nephritis is associated with infection caused by certain specific serologic types of streptococci.

Ever since the group A streptococci were first classified into some 40-odd types, a number of attempts have been made to attach special clinical significance to specific types. For example, for a time it was thought that only certain serologic types of streptococci could produce scarlet fever, but it was subsequently shown that almost all types have been associated with this disease at one time or another, and that the appearance of the rash is related to the erythrogenic toxin produced by the strain and not to the type. Also it has been proposed that certain types of group A streptococci may be rheumatogenic or nephritogenic.

It is difficult to obtain attack rates for rheumatic fever and acute nephritis following known types of streptococcal infections. Even in many university hospitals typing of streptococci is not a routine procedure, so that often the type is unknown. In addition, one is likely to get striking differences in attack rates merely on the basis of definition of what is and is not streptococcal infection, and of what is or is not rheumatic fever or acute nephritis. Furthermore, it is hazardous to compare attack rates obtained at different times or in different places.

However, evidence has been carefully collected on attack rates for both rheumatic fever[12 26] and acute nephritis[11 25 27] following well documented streptococcal infections of different serologic types. These data have shown a remarkably constant attack rate for rheumatic fever, regardless of the type of streptococcus involved. In contrast, the attack rate for acute nephritis varies strikingly with the type of streptococcus which initiated the preceding infection. Following infection with most types of streptococci, virtually no acute nephritis develops. Following other types the attack rate is as high as 18 per cent. Types 12, 4, 25, and 49 are especially likely to result in kidney damage. Type 49 is a new serologic type first identified on the Red Lake Indian reservation[28] and in family outbreaks in Minnesota[29].

Nephritogenic strains

The demonstration by Rammelkamp and colleagues that certain types of streptococci are nephritogenic explains many of the clinical and epidemiologic differences of acute rheumatic fever and acute nephritis. For example, it is now apparent why thousands of troops had nephritis during World War I, whereas—even in the face of pandemic streptococcal disease—little or none was seen in the military during World War II. Thus the occurrence of nephritis is not related to the presence or absence of streptococcal infections *per se*, but rather to the presence or absence of infections with specific *nephritogenic* strains.

These findings also explain why, in contrast to rheumatic fever, patients rarely have a second attack of acute hemorrhagic nephritis. Since immunity to streptococcal infection is type-specific, patients who have acute nephritis resulting from infection with a nephritogenic type will be subsequently immune to that type[5], and are not be likely to be infected with the few remaining nephritogenic types.

It also explains why in a family outbreak of streptococcal infection several members may develop acute nephritis simultaneously. This may occur whenever a nephritogenic strain is introduced into a family.

Unexplained differences

Certain other differences between the two diseases are not yet clarified by the identification of nephritogenic strains of streptococci. The significance of the difference in geographic distribution is still obscure. Although differences in geographic distribution of certain streptococcal strains have been described, the nephritogenic types have apparently been widely distributed over the United States in recent years. Also, the association of deep infections, and more particularly of skin infections, with acute nephritis remains unexplained.

Although skin infections are frequently reported in connection with acute nephritis, the interpretation of cultures of skin lesions is difficult. Often the hemolytic streptococcus is overgrown by staphylococci, in which case its presence can be detected only by meticulous bacteriologic studies. When streptococci are isolated from skin lesions, they can almost always be isolated from the upper respiratory tract as well. This makes it hard to decide which was the original site of infection, and which site is important in the etiology of nephritis. The possibility that those strains or types of streptococci which lead to nephritis have a special propensity for infecting the skin must be considered. It is also possible that the staphylococci which secondarily infect the skin may contribute to the pathogenesis of nephritis by some symbiotic action with streptococci.

Epidemiologic evidence that only a limited number of types of streptococci can produce nephritis indicates that these strains must have in common some biologic property which is lacking or relatively deficient in most strains. Bernheimer and others[30] have recently found that nephritogenic types of streptococci tend to produce large amounts of an enzyme which splits DPN. The finding in our laboratory that patients with nephritis tend to show higher titers of neutralizing antibody for DPNase than patients with rheumatic fever[31] suggests that this enzyme should be studied more thoroughly for its possible role in the etiology of acute nephritis.

Clinical Implications

Apart from the interesting theoretical aspects of the similarities and differences in the epidemiology of acute rheumatic fever and acute nephritis, there are definite clinical implications which bear on the diagnosis, management, prognosis, and prevention of these diseases. After a streptococcal infection the physician should follow the patient carefully, looking for manifestations of kidney or heart disease, such as hematuria or the development of murmurs. Whenever a history of a sore throat or a skin infection is obtained, these two diseases should be kept in mind. Skin infections or deep infections should suggest particularly the possibility of acute nephritis.

Unfortunately, there is no specific laboratory test for the diagnosis of either disease, but in problematic cases it is helpful to look for high levels of streptococcal antibodies. For example, an antistreptolysin O (ASO) titer of more than 250 units suggests that the patient has had a recent streptococcal infection, and is circumstantial evidence supporting the clinical impression. Since approximately 20 per cent of patients with streptococcal infection fail to show a rise in ASO titer, antibody tests for other streptococcal antigens should be performed when the ASO titer is borderline or low[2b]. High anti-DPNase titers are particularly common in acute nephritis[31]. It is also possible to look for specific antibodies for the nephritogenic types of streptococci[2b], but the latter procedure is not suitable for routine diagnostic laboratories.

Prognosis

There are several implications concerning prognosis. A patient who has had an attack of rheumatic fever is very likely to have a recurrence if he has another streptococcal infection. This does not appear to be true of acute nephritis. Following an attack of the latter, the patient is immune to the nephritogenic type of infection that preceded it and is unlikely to become infected with the few remaining types which might lead to a second attack.

The prognosis of acute nephritis is excellent, especially in children or in epidemics among adults. Some investigators believe that acute nephritis following known streptococcal infection in an individual without previous kidney disease rarely, if ever, leads to chronic nephritis[8]. In their view, the etiology and prognosis of the two conditions are quite different. This point is still controversial[32], and the difficulty in resolving it is apparently related to the difficulty in distinguishing between an attack of acute nephritis and a streptococcal exacerbation of a previously undiagnosed case of chronic nephritis[8]. The problem is particularly knotty in adults, who are more likely to have pre-existing renal disease. A favorable outcome can be anticipated in children who have hematuria following streptococcal infection, since in this age group almost all cases of chronic nephritis develop insidiously, with no acute episode of gross hematuria.

Management and prevention

The implications concerning management and prevention are clear and important. Streptococcal infections of the throat or skin should be treated with penicillin. In order to prevent rheumatic fever, the infecting streptococcus must be eradicated[33]. The dosage, the route of administration, and the kind of penicillin are relatively unimportant. On the other hand, the duration of effective treatment is most important. Penicillin must be administered in such a fashion as to maintain blood levels for 10 days[34]. Intramuscular administration of benzathine penicillin (Bicillin) is a convenient method of assuring adequate duration of treatment. Oral penicillin may be prescribed, but it is difficult to get patients to continue it after the symptoms of acute infection have subsided.

Early treatment of infections caused by nephritogenic strains of streptococci will reduce the incidence of acute nephritis, but penicillin appears to be less effective than in the prevention of acute rheumatic fever. In particular, treatment begun late apparently fails to prevent acute nephritis[25], but when delayed as much as nine days will significantly reduce the incidence of acute rheumatic fever[21a]. The relative ineffectiveness of penicillin in preventing acute nephritis, including the prophylactic failure of late treatment, may be related to the shorter latent period of the disease and supports the possibility that some streptococcal product exerts a direct toxic effect in the pathogenesis of this disease. The prophylactic value of eradicating the infecting organism in acute nephritis has not been established, but for the prevention of acute rheumatic fever it is advisable to obtain follow-up throat cultures to make sure that the organism has been eradicated, especially in patients being treated with oral antibiotics.

If acute nephritis is diagnosed in one member of a family or in one child in a classroom, it is advisable to look for evidence of this disease or of streptococcal infection in other members of the family or school. The recognition and treatment of other cases of clinical or subclinical infection may halt further spread of the nephritogenic strain and thereby prevent the occurrence of an epidemic of nephritis.

A patient with acute rheumatic fever should be given a therapeutic course of penicillin to eradicate any group A streptococci which may be present in the throat. He should then be placed on prophylactic penicillin. A monthly injection of benzathine penicillin (Bicillin) or an oral dose of 200,000 to 250,000 units once or twice daily appears to be adequate[34]. In uncooperative patients, the long-acting intramuscular preparation is preferable. The length of time that prophylaxis should be continued has not been established with certainty. The greatest risk extends through adolescence, although young men in military service and mothers of school-age children are also vulnerable. Because some risk continues for at least 20 years[35], it is recommended at present that prophylaxis be continued indefinitely[34].

In contrast to rheumatic fever, second attacks of acute nephritis are so rare that there seems to be no logic in placing such patients on long-term continuous prophylaxis. Some physicians elect to continue prophylaxis for several months to one year while the Addis count is still normal, since streptococcal as well as non-streptococcal infections may cause a temporary increase in hematuria during this period. The possible benefit of this procedure is difficult to evaluate because of the high incidence of complete recovery when no prophylaxis is given. Indeed it can be argued that patients with acute nephritis should not be given penicillin therapeutically or prophylactically, as these procedures may inhibit the development of type-specific antibody[36], which protects them against subsequent infection with this nephritogenic strain.

Summary

Many of the differences in the clinical and epidemiologic features of acute rheumatic fever and acute nephritis may be explained by the existence of nephritogenic strains of streptococci. Studies of the role of streptococcal infection in these diseases have not only contributed to our understanding of their behavior, but have suggested practical methods for their management and control. In addition to the intriguing theoretic aspects, the epidemiologic differences of the two diseases have important clinical implications concerning the treatment, prevention, and prognosis of streptococcal infections and their complications.

A complete list of references will be included in the author's reprints.

It cannot be denied—the record demonstrates it incontrovertibly—that social concepts and programs which originally were embraced to help people to help themselves—and thus to preserve in them a spirit of independence and self-reliance — have been progressively distorted and modified so as to thwart and stifle those saving attributes in our citizens and to develop in them instead a despicable eagerness to depend and rely upon government.

Philosophic Implications of Terminal Illness

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Few decisions in medicine can be made without thoughtful consideration of alternatives. The proper balance of the science and art of medicine often requires a wisdom greater than we can muster. The physician's role implies scrupulous attention to minute detail, and intimate knowledge of and appreciation for the economic, social, moral, pathologic, and physiologic aspects of humanity.

Ethical standards are not acquired by repeating the Oath of Hippocrates or the Prayer of Maimonides, nor is a moral sense developed by osmosis alone on the hospital wards. Though physicians, we must acquaint ourselves with the discussions of social scientists and philosophers. The physician must hold to his Hippocratic oath, but in the light of present day knowledge and his own spiritual values.

Many children with a terminal illness are referred to the Pediatric Service at Duke Hospital. The experience of taking care of young patients during their final days and of deciding how they should be managed furnishes the impetus for introspection and soul-searching on the part of the physician. He finds himself in philosophic discussions with his colleagues. He avoids certain aspects of the problem of terminal care and faces others. My experiences on Howland Ward at the Duke Hospital made me aware of the complexity of this aspect of medical practice. I have pondered its psychologic and philosophic implications, as have most physicians.

In the management of terminal or catastrophic illness there is no black and white pattern which we can adopt and follow rigidly with each patient. Wherein lies the glory of a technical triumph which precipitates economic, social, or spiritual bankruptcy? When faced with complicated medical problems we cannot allow culpable ignorance to mask itself in the guise of humanitarianism; neither can we allow scientific achievement to preclude the right to live or to die with the dignity which is the right of every man.

Prolonging Life or the Act of Dying?

After George Washington, dying of edema of the larynx, had suffered repeated bleedings, purgings, and blisterings—then current methods of scientific medicine—he implored his persecutors: "I pray you to take no more trouble for me. Let me go quietly."

George Washington's physicians were diligent to the end because in his case they thought they were prolonging life, but actually they were prolonging the act of dying. With our modern scientific skills and drugs we can prolong the act of dying to frightening lengths. Often we hear statements today, especially regarding the elderly, that science will not let people die naturally, even when they are ready and longing for death. This attitude is often considered respect for life, but one wonders if it is not, in the words of Martin Buber, "man's lust for whittling away the secret of death." The good physician trains himself to prolong life, but not the act of dying. The terrifying dilemma which often confronts us is "which are we doing?"

If it has been established beyond any doubt that the patient is *in extremis*, if the physician and his associates are in unanimous agreement, and if there is no question in anyone's mind about the prognosis, are extraordinary measures indicated to keep the patient alive? If neither the patient nor the relatives express a wish for heroic measures, then who does ask that they be used? Is it the patient's church? No voices representative of the Roman Catholic or Greek Orthodox Churches or of the Jewish or Protestant faiths have suggested that physicians should try extraordinary means to keep life going when every process of the body is determined to die. His Holiness, Pope Pius XII, issued an encyclical during the last year of his life which stated the official position of the Roman Catholic Church as not requiring extraordinary means when only certain death and suffering lie ahead.

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The meaning of the term "extraordinary" is discussed in textbooks of medical ethics by Catholic authors as whatever here and now is very costly, unusual, painful, difficult, and dangerous, or if the good effects that can be expected from its use are not proportionate to the difficulty and inconvenience that are entailed. This has reference, of course, to the patient whom the physician and his consultants have sincerely judged incurable. Since the professional standards of conscientious physicians vary somewhat regarding the use of artificial life-sustainers, the physician should feel free in conscience to use or not use these measures according to the circumstances of each case. The Roman Catholic Church also recognizes that, by reason of certain special conditions, failure to use extraordinary means may reflect unfavorably on the medical profession. In such a circumstance the physician may not feel free in conscience to follow his best medical judgement.

Errors in Judgment

The difficult dilemma often facing the physician is whether his competence and clinical judgment are always sufficient to distinguish between prolonging life and prolonging the act of dying. If he can make this distinction with certainty, then the course of action seems clear cut. At times he will make errors in judgment, and these will haunt him, though possibly they should not.

In an informal discussion on this subject, a brilliant surgeon told of a young man with difficulty in swallowing who was admitted to the ward on which the speaker was then a surgical resident. The patient was studied thoroughly and was diagnosed as having carcinoma of the esophagus. The x-ray findings revealed a fistula through which some of the radio-opaque medium had spilled into the lungs. The staff decided that he had an inoperable lesion and that therefore the only treatment indicated was supportive care which would keep the patient comfortable as he slowly died. The patient did eventually die. The postmortem examination revealed an emaciated human being, but absolutely no carcinoma of the esophagus. The shadows on the x-ray films which had been interpreted as evidence of cancer and the fistula turned out to be artifacts. The patient actually had a psychogenic illness, possibly anorexia nervosa. The error in judgment led to the wrong therapeutic regimen, about which the surgeon still feels guilty.

The Tendency to Withdraw

Usually, however, the physician is able to distinguish between prolonging life and prolonging the act of dying. If he decides that the case is terminal, and that he must not interfere with the act of dying but must give nature a freer hand in the outcome of the illness, he should guard against abandoning his patient as death approaches. An experience during my junior year in medical school dramatically brought to my attention the tendency to withdraw from a dying patient.

One of my classmates was assigned to take care of a 30 year old white man with leukemia. Our clinical group of three students knew this patient and his wife, and participated in his care for the three months he was in the hospital prior to his death.

After treating the patient vigorously for about two months, the resident and attending staff suggested that treatment be withdrawn, for at this point it seemed that we were prolonging not life but the act of dying. We (the students) did not understand how the treatment could be relaxed, and discussed among ourselves the ethical implications of such a course of action. The staff sympathized with our need to continue treatment and gave us a fairly free hand in the patient's management. On the ward was a patient with polycythemia vera whose blood matched that of the leukemic patient. We drew his excess blood and used it for transfusing the other. Finally we became certain that the leukemic patient was going to die. Then the two of us who were not responsible for his care began to stay away from his bedside, whereas previously we had been regular attendants in his room, mostly for conversation with him and his wife. We were hardly aware that we were staying away. One day I met his wife in the hall, and she asked, "Why don't you come to visit us any more?" At a loss for words, I finally muttered, "Well, since we can't do anything, we would only be in the way." She replied, "Oh, no; he misses you. He knows he is not going to live and that medication will not help him, but both he and I want to share with you our thoughts and feelings in this crisis. You could be of great help to us."

The Physician an Instrument of Therapy

This experience gave our group an opportunity to explore with the staff several crucial factors in the care of terminal patients. In the first place, the physician must never overlook himself as an instrument of therapy. Mankind has known from the beginning of time that healing and comfort come from human relationships. The physician forgets this sometimes in his emphasis on drugs and surgical procedures. Second, why did we stay away? Two of us could rationalize that we were not responsible for this patient's care, but both he and his wife looked upon all three of us as active in the treatment process. Possibly it was because of our anxiety about death. A number of medical students and physicians have not worked through their own feelings about death and the finiteness of their own lives. Thus they cannot be comfortable with a patient as he approaches death. Guilt feelings must also be reckoned with. On some level of awareness we realized that a man was dying while we were allowed to live. Why? Another major possibility was that this death was an attack on our "fantasied omnipotence." We were losing a battle with death, and we felt impotent. Death had been presented by our instructors as the great enemy of the physician. When a fight with death is lost, the physician is confronted by feelings of inadequacy as a healer.

Our instructors insisted that regardless of how we feel, we should never abandon the dying patient, especially if we are at all responsible for his care. We must be willing to discuss with him any concerns which may be brought up in those final days.

The dying patient experiences a penetrating type of loneliness and aloneness. He needs a shoulder to lean on as he becomes oriented to his new situation and works out some kind of solution to this approaching fate. The family also has its burdens and needs the physician. If physician, family, and friends form a true community of spirit with the dying patient, the feelings of isolation and separation accompanying death are neutralized, or at least minimized.

Such was the situation with a 5 year old boy who suspected from the tragic expression on the faces of his parents that he was going to die. He asked one of his doctors if this were the case. His doctor told him honestly that he was very sick and that so far the treatment had not been very effective. He asked the boy gently if he were afraid, and the boy returned the question. They both decided that they were not. The following day when the boy's parents visited him on Howland Ward, he told them that everything would be all right regardless of what happened, and that they should not worry. They asked, "Why do you feel that way?" His simple and moving reply was, "Because my doctors love me."

Conclusion

Terminal care tests more than the medical skill of the physician. It calls for the subtle blending of the art and science of medicine to such a refined degree that, even in death, victory and honor are achieved by both patient and physician. Walt Whitman challenges us through his words: "The faithful hand of the living does not desert the hand of the dying."

Management of Pyelonephritis in Childhood

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Pyelonephritis is the most common renal disease and the primary cause of renal failure in infants and children. An incidence ranging from 15 to 20 per cent is found in general hospital autopsies[1]. However, more rigid criteria for a microscopic diagnosis of the disease have been suggested and may lead to a significant reduction in these figures[2]. Thorough discussions of clinical and experimental pyelonephritis are available in recent reviews[3].

Pathogenesis

The high incidence of this infection in infancy and childhood is marked by its predominance in the female—2:1 in the first year and 6:1 thereafter. The most important factor in establishing the infection is any process, either congenital or acquired, which impedes the natural flow of urine. It is estimated that chronic urinary tract infection is 20 times more frequent in children with urinary tract anomalies than in normal children, and that the obstructive type of pyelonephritis is 12 times as frequent as the non-obstructive type[4]. The significance of these figures is emphasized by the finding that 12 per cent of all newborn children demonstrate some urogenital anomaly[4b].

Bacterial inoculation of the kidney can occur by way of the blood stream, by direct ascent from the bladder, by spread from contiguous tissues, and possibly through lymphatic channels. Gram-negative bacteria, predominantly the coliform group, account for 80 to 90 per cent of these infections[5]. Because the normal distal urethra contains mostly gram-negative bacteria and the majority of patients are female, it is understood why this form of bacterial pyelonephritis is considered secondary to infection ascending from the lower tract. However, instrumentation of the urethra and bladder can cause a gram-negative bacteremia which, under certain circumstances, could cause an acute infection of the renal parenchyma[6]. Hematogenous inoculation also occurs in the neonatal period, when gram-negative septicemia is most commonly noted. Overgrowth of these bacteria may take place in the normally gram-postive flora of the respiratory tract in patients receiving antibiotics. This not only affords another site of entry into the blood stream, but also largely by-passes the very important trapping effect of the liver in the initial circulation to the kidneys.

Except in the neonatal period these coliform bacilli are of a lower order of virulence for most body tissues. They are, however, the commonest cause of renal infections, and the kidney apparently has considerable difficulty in eradicating them. This has been noted also in experimental animals. After the intravenous injection of certain bacteria it was observed that the kidneys removed only a small portion of the inoculum and that the liver and spleen trapped the major part. Complete destruction of the bacteria in the liver and spleen occurred subsequently in some animals, in contrast to the persistence of the live organisms in the kidney[7]. Investigation of this difference between organs has demonstrated that the ammonia formed in the kidney inactivates the complement. thereby interfering with one of the mechanisms involved in the eradication of *Escherichia coli*[8].

It is speculated that in acidotic conditions which increase the production of renal ammonia, sufficient complement-inactivation may occur in some parts of the kidney to allow bacterial infection to become established. Such a mechanism was considered in diabetes mellitus, where pyelonephritis is both common and severe[8]. It might also be involved in infants with severe diarrheal acidosis, where the incidence of urinary tract infection is higher[9]. Not only is the infant's urethral meatus bathed in contaminating stools, but the severe dehydration causes a marked oliguria. Severe oliguria might be considered a state of "functional

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obstruction" in the urinary tract causing a transient stasis and thus facilitating the ascent of bacteria to the kidney. If the above mentioned mechanism does operate in the human, the organisms would seem to have an optimal environment for establishing infection. Contemporary management consisting of efficient correction of dehydration and acidosis and frequent use of antibiotics has apparently brought about a decrease in these secondary infections. Which factor or combination of factors is responsible for preventing this complication is yet to be determined.

The remaining 10 to 20 per cent of acute infections are caused by the various coccal organisms, the staphylococci being the most common, the *Streptococcus fecalis* (enterococci) the least common, and the beta and alpha streptococci in between. These infections are usually secondary to spread by the blood stream from a distant focus—for example, staphylococci from a carbuncle. Any of these organisms, however, can be found in a normal urethra, and infection from direct ascent or secondary to instrumentation is a possibility.

Bloodborne staphylococci usually concentrate in the cortical areas as multiple suppurative foci. Coalescence may result in a renal carbuncle. Outward penetration leads to perinephric abscess. Superior extension could cause pleuritis, pneumonia, and even lung abscess, whereas downward extension along the ureter may cause a pelvic abscess. Such extension and complications are uncommon during the invasion of the urinary tract by coliform bacteria, because the extrarenal bodily defenses are usually capable of eradicating these less virulent organisms from the other organs and systems.

Catheterization

The role of the catheter in the etiology of urinary tract infections has been the subject of much discussion[5a]. Even with strict aseptic techniques, the incidence of such infections following catheterization is reported as 4 per cent[10]. Catheterizing patients with preexistent infection is reported to increase the danger of enterococcal infection[11]. Actually, a properly performed catheterization carries minimal risk. The procedure is considered hazardous because it is frequently performed by inexperienced personnel or is attempted without help. The latter is especially likely to be true when the patient is an uncooperative child. Not only does the field become easily contaminated, but the urethral and bladder tissues can be injured. If the distal urethral organisms are carried in with the catheter, the traumatized area can become infected. In the light\of this knowledge, it would seem that only the most experienced and capable personnel should be responsible for this potentially dangerous procedure.

With indwelling catheters, infection is almost inevitable, and is not prevented by irrigation and antibiotics[12]. In fact, some resistant organisms may be superimposed in patients receiving prophylactic antibiotics[13].

Course and Recognition

A sudden onset of high fever, chills (possibly convulsions or meningismus in infants), flank pain and tenderness associated with pyuria and bacteriuria seldom offers difficulty in recognizing pyelonephritis. The onset is frequently preceded by acute cystitis manifested abruptly by increased frequency of urination, dysuria, and suprapubic pain. Severe bladder inflammation may lead to bleeding or hemorrhagic cystitis. Most of these infections respond to appropriate drug treatment if no obstructive uropathy exists.

In about 30 per cent of patients with chronic pyelonephritis, no such clinical signs and symptoms are present. The clinical picture may be atypical or even asymptomatic, especially in infancy. This fact is well documented in a report of 85 cases of pyelonephritis in children under the age of 2 years[14]. Only one case was correctly diagnosed prior to hospitalization. The diagnosis is easily missed because the signs and symptoms may be predominantly gastrointestinal, the physical findings minimal or normal, and the urinalysis insignificant or normal. In this same report a symptomatic onset was noted in the first year of life in 91 of 156 chronic cases in children. Anorexia and vomiting were the dominant symptoms, and failure to gain weight, irritability and constipation were present in the large majority. Changes in micturition were elicited only by direct questioning in about one half of the cases, frequency being the most common change recorded.

The state generally reported as "failure to thrive" may not be present. Except for intermittent periods of irritability associated with one to three days of temperatures ranging from 101 to 103 F., the infant's growth, development, and behavior may appear normal. Routine examination during these episodes is either negative, or a "pink throat" is observed, for which two to three days of an antibiotic is often prescribed. The antibiotics may not only temporarily suppress the urinary tract infection and mask other clinical signs or symptoms, but provide the setting for the emergence of more resistant forms of bacteria. Not infrequently these episodes are ascribed to "teething." Any infant who manifests fever with so-called "teething" should have a properly performed urinalysis, including smear and culture for bacteria.

Although some patients may recover without treatment, the majority suffer the eventual effects of smoldering and relentless renal disease manifested by chronic ill health, recurrent febrile disease, and death from renal failure, with or without hypertension.

In general, the large majority of these patients have a significant pyuria, about two thirds have proteinuria ranging from a trace to 1 plus (rarely exceeding 1 to 2 Gm. per 24 hours in acute disease or 3 Gm. per 24 hours in chronic cases[3b]), and about one half have increased numbers of red blood cells (rarely, gross blood) in the urine. Only freshly voided clean-catch urine should be examined. The microscopic examination is performed on uncentrifuged specimens, and more than two white blood cells per low power field is considered significant[15]. Such values are unreliable, since the concentration of the urine as well as the time of collection causes marked variations. Furthermore pyuria can be associated with extrarenal infections[16]. Clumps of white cells usually signify active infection, but mucus from contaminating vaginal secretions can also cause clumping. Pale-staining leukocytes prepared with a supravital stain are noted in other diseases involving parenchyma, but their presence is very helpful in differentiating pyelonephritis and ureterocystitis[17].

More significant is a positive smear for bacteria in the uncentrifuged urine. A positive gram-stain preparation correlates well with significant bacteriuria (greater than 100,000 bacteria per milliliter of urine)[18]. For both culture and routine examination the properly collected, clean-catch, midstream sample is reliable for most cases of suspected infection[18].

Unfortunately, a routine urinalysis my be negative even during active infection. This may be due to failure of the inflammatory process to rupture into the renal tubules at that stage. Also, cases of unilateral pyelonephritis with transient ureteral obstruction could be associated with a negative urinalysis. More frequently, however, the microscopic examination is normal, but the bacterial smear and culture are positive. When an organism fails to grow in significant numbers or to grow at all in artificial media, the direct smear examination would be more reliable. Occasionally the quantitative culture consistently reveals organisms in the contamination range, but clinically the case appears as an indolent form of disease. In order to clarify this problem as being either a low-grade infection or urethral contamination, resort to a direct suprapubic needle puncture of the bladder for urine culture may be necessary[19].

Renal biopsy has been of considerable help in patients where it is difficult to differentiate by routine tests an indolent latent pyelonephritis from a latent nephritis or nephrosis, or where collagen processes with renal involvement may be present. Although a routine biopsy can miss the area of pyelonephritic involvement, the pathologic evidence of the other processes, if present, is usually apparent on microscopic study.

Management

Appropriate drug treatment should be

started as soon as the diagnosis is confirmed by gram stain and/or culture. A methylene blue stain is acceptable if gram stain is unavailable. Since 80-90 per cent of the cases of acute urethritis, cystitis and pyelonephritis in children are due to *E. coli,* and since the large majority of these infections are amenable to sulfonamides, these drugs are initially the preferred therapy. A triple sulfonamide or sulfisoxazole (Gantrisin) is started in the usual dosage, and if a good clinical response is noted within 24 to 48 hours, is continued for 10 to 14 days. A favorable response cannot be judged by clinical course or routine urinalysis alone. About two or three days after drug therapy is discontinued, a repeat stained smear and if possible a culture are obtained. If these tests are normal and the patient remains asymptomatic, they should be repeated again three and six months later. If they are negative the condition is considered cured.

If the initial clinical response is poor or if bacteria persist on either routine smear and/or culture, another drug such as a tetracycline should be started (Chlortetracycline is the most active of this group of drugs against most susceptible bacteria) and the program continued as outlined above. If a resistant *E. coli* is suspected or proved, a combination of streptomycin (7 days) and tetracycline (14 days) is advisable. Drugs such as kanomycin are used if all others have failed.

If the patient is a male, even if he has an acute disease—respiratory or intestinal, for example—a urologic survey for renal anomalies should be performed after the acute infection is controlled. If the patient is a female, and especially if she has an acute diarrhea, a urologic survey may be omitted at this time, but must be done if the infection persists or recurs.

If the pathogen is found to be the *Aerobacter aerogenes,* Proteus or paracolon groups, initial treatment with streptomycin (7 days) and tetracycline (14 days) is advisable. Some of the Proteus species are susceptible to sulfonamide drugs and others to high doses of penicillin. *Pseudomonas aeroginosa (Ps. pyocaneus)* requires drugs such as polymyxin or the newer colymycin for therapy. Sensitivity studies are of value, especially when a more resistant organism is encountered.

Acute urinary tract infection associated with a focus of staphylococci and gram-positive cocci in the urine is treated with penicillin, if considered non-resistant, or Ilosone if the patient is sensitive to penicillin. If response is poor in 24-48 hours, a more potent drug such as Chloromycetin is indicated. The original focus should receive local treatment, such as drainage, when indicated. Urologic survey is not indicated in this gram-positive, hematogenous-spread group of infections, especially if the response is complete and follow-up examination continues normal. If infection is caused by the enterococcus, a combination of penicillin and streptomycin is the treatment of choice.

Occasionally a problem arises with the young female whose initial disorder is urethritis-cystitis which clears completely, but recurs two to three times in the next 18-24 months, and in whom no urologic abnormalities are noted. If vaginitis or other local irritation (frequently secondary to pinworm infestation) is observed, questioning of the mother usually reveals improper hygiene techniques after urination and defecation. Mothers should be instructed on post-urination "blotting" (rather than wiping) and a posterior anal wipe. Shower baths rather than tub baths should be encouraged and summer wading pools avoided.

Any infant or child found to have pyuria or proteinuria on a routine check and bacteriuria upon further examination should have a thorough urologic investigation and be placed on drug therapy. Anomalies should be corrected when feasible. Even sensitive organisms such as most of the *E. coli* are difficult to eradicate if any condition is interfering with urine drainage, such as incomplete obstruction or ureterovesico reflux. Long term therapy (12-18 months) is required in stubborn cases, but judicious use of available drugs and urologic procedures is essential if the indolent process is to be arrested. Once chronic pyelonephritis is established, even without recognizable obstructions or malfunction of the urinary tract, only about 10-15 per cent of the cases can be cured with contemporary management[13].

Summary

Pyelonephritis as a childhood infection ranks only behind respiratory and gastrointestinal diseases in frequency. The disease is frequently associated with anomalies of the urinary tract, some of which are extremely difficult to demonstrate. Both the anomalies and the disease may exist singly or together without producing signs and symptoms, or without their usual manifestations, until irreversible renal damage has occurred. Thus the physician must be alert for these conditions in any infant or child who has unexplained fever or who is not progressing normally, regardless of the presenting complaints.

Treatment of the infection cannot be based on clinical response, and to be considered successful should result in negative bacterial smears and/or cultures at least three and six months after treatment is completed.

A complete list of references will be included in the author's reprints.

Some Syndromes Produced by the Toxin of Insects

George E. Prince, M. D.
Gastonia

During the summer months I see many patients who have been bitten or stung by insects. Some insects produce syndromes having specific diagnostic features. Four such syndromes are presented in this paper.

Stings of the Puss Caterpillar

For the past two years, in late September or early October, I have treated patients who had been stung by the puss caterpillar, *Megalopyge opercularis*. The caterpillar is the larval stage of the flannel moth. It is easy to identify because of the fur-like quality of the dorsal setae, or hairs, and by the fact that the hairs are longer over the last two segments of the worm, producing a conspicuous tail (fig. 1).

The puss caterpillar has two types of setae (fig. 2), one of which contains many bristles and is readily detachable. It has been the cause of some cases of conjunctivitis and bronchitis. The other type is smooth and sharp and contains a venom which is responsible for the toxic signs and symptoms. The venon-containing setae are located beneath the furry ones, and will break off in the victim's epidermis. The severity of the resulting symptoms has some correlation with the size of the caterpillar and the amount of pressure exerted on contact.

Intense shooting pains usually occur within 10 or 15 minutes of the sting. Itching and localized swelling follow, and erythema usually develops around the site. Intense pain is noted also in the axilla or groin, if an extremity is involved. Regional lymphadenopathy occurs in more than half of the victims, and some become nauseated and vomit. All my patients became emotionally upset and extremely apprehensive. McGovern and others[1] have reported 5 instances of convulsive seizures in a series of more than 2,000 cases.

The onset of symptoms was so rapid in each of my cases that the caterpillar was found and brought to the office with the child.

I have personally experienced the sting of this larva. The pain was terrifying and lasted several hours. Foot[2], in 1922, performed experiments on himself and others showing that the venom diminishes in virulence after the larva has spun its cocoon, and is no longer active after the caterpillar is dead. I have produced regional lymphadenopathy by rubbing the dry setae into the skin, but no pain or other symptoms were noted. The setae were still capable of producing lymph node enlargement seven months after the caterpillar had died.

The best treatment seems to be the use of Demerol or other analgesics for the relief of pain, and intravenous injections of calcium gluconate when severe generalized reactions

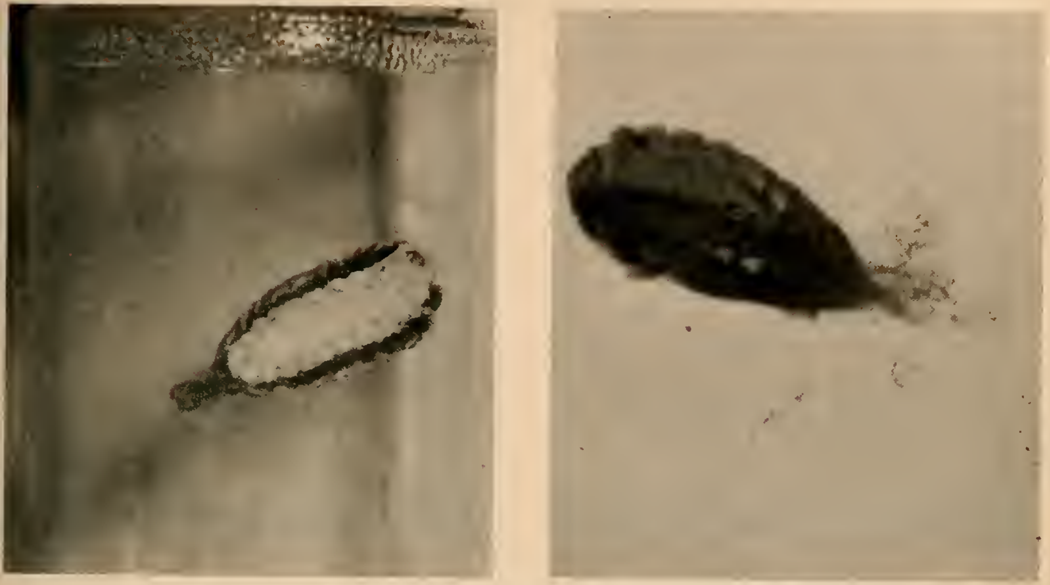

Fig. 1. The puss caterpillar. Left, dorsal view. Right, ventral view.

occur. Epinephrine, antihistamines, and adrenal steroids did not appear to influence the clinical course. Most of the symptoms will disappear within a few hours even if no treatment is given.

Tick Paralysis

Another of the syndromes attributable to insects is tick paralysis, one of the easiest and most gratifying to diagnose. It is caused by a toxin produced in the salivary glands of the female tick and injected into the patient during the period of engorgement. In the United States the species responsible for paralysis are the common dog tick (*Dermacentor variabilis*), the wood tick (*Dermacentor andersoni*), and the Lone Star tick (*Amblyomma americanum*). Recently it has been demonstrated that the toxin blocks conduction in somatic motor nerve fibers, thus inhibiting the release of acetylcholine at the neuromuscular junction. The resultant paralysis is similar to that seen in botulism.

The patient with tick paralysis is usually a young girl who suddenly becomes irritable and weak. Ataxia and loss of tendon reflexes are next noted. The paralysis which develops is flaccid, symmetrical and painless. As it ascends, the muscles of deglutition and respiration may be affected. No signs of meningeal irritation are found, and the spinal fluid, hemogram, temperature and pulse are normal.

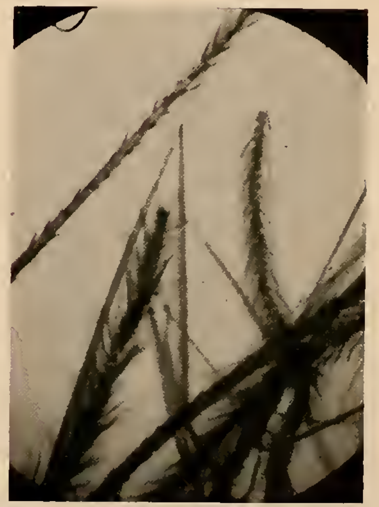

Fig. 2. Setae of the puss caterpillar as seen under the microscope. The smooth ones contain venom.

Treatment consist of removing the tick and its mouth parts from the patient. In nearly all cases dramatic improvement occurs after this is done, and most of the weakness and paralysis disappear within 48 hours. In many of the reported cases the tick has been found accidentally, and frequently not until the patient has been sick several days.

Supportive treatment includes the use of oxygen, parenteral fluids, suction, postural drainage, and at times a respirator. The reported mortality is about 4 per cent. The disease can best be prevented by the daily inspection of children for attached ticks and by carefully removing any that are found.

There are several theories as to why this disease is rare when tick infestation is common. Some investigators believe that only pregnant female ticks containing fertile eggs are capable of producing the toxin. In nearly all reported cases the tick has been a female, but the state of fertility of the eggs has not been determined. It is possible that most ticks are removed from children before enough toxin is injected to cause paralysis.

A female dog tick, *Dermacentor variabilis,* was removed from one of my patients who had tick paralysis, and about 3.000 of the eggs were ground and suspended in saline[4]. This suspension was injected into a white mouse. The mouse became paralyzed and died within 26 hours. Blood was taken from the heart and 0.5 cc. was injected subcutaneously into another mouse. Local irritation was observed, but no paralysis developed. The tick died when placed in a rabbit. The eggs not used in the suspension were kept at room temperature for one month, but did not hatch. The period of observation was too short, since it often takes as long as seven weeks for the eggs to hatch.

When examining a patient who has flaccid paralysis, no signs of meningeal irritation, and a negative spinal fluid, one should make a thorough search for engorged ticks.

Black Widow Spider Poisoning

Poisoning by the black widow spider (*Latrodectus mactans*), or arachnidism, is not uncommon in North Carolina[5]. Most practitioners in the state have treated it.

This spider is not aggressive, and attacks only when it is disturbed. The venom injected is a hemolysin; it also stimulates the myoneural junctions and acts on nerves and nerve endings. Not all persons who are bitten become sick, and there is evidence that some individuals are immune to the venom. There is little local pain or reaction, and in only 10 per cent of the cases has the spider been found on the patient.

The sequence of symptoms is usually the same no matter where the venom is injected, although cramps may begin in the skeletal muscles nearest the site. The symptoms develop within 15 to 30 minutes of the bite, and the chief complaint is severe abdominal pain. Vomiting and headache are common.

When the patient is examined shortly after the onset of symptoms, he is often rolling restlessly from side to side. If he is a child, he may be uttering expiratory grunts, and his legs may be drawn up against the abdomen. The abdominal wall becomes rigid and tender, but no localized or deep tenderness is present. The abdominal reflexes are diminished or absent, and the tendon reflexes are hyperactive. The body temperature and blood pressure are frequently elevated. The white blood cell count may be normal or above 20,000 per cubic millimeter. The urine often contains albumin and occult blood.

Treatment, when indicated, is specific. Ten cubic centimeters of a 10% solution of calcium gluconate is given intravenously. The first few cubic centimeters are given rapidly to produce a flush or drowsiness. The remainder of the dose is given slowly. Dramatic relief of pain is obtained within five minutes. An intradermal skin test for horse serum sensitivity is then done. If the result is negative, 2.5 cc. of antivenin (Lyovac, *Latrodectus mactans*) is injected intramuscularly. Both calcium gluconate and antivenin may have to be given again in a few hours if symptoms return or persist. I have given ACTH and cortisone when the patient was found to be sensitive to horse serum. Improvement was slower in the patient not receiving the antivenin.

Recently a case of arachnidism treated with methocarbamol (Robaxin) has been reported[6]. Since antivenin and calcium gluconate were administered first, it was

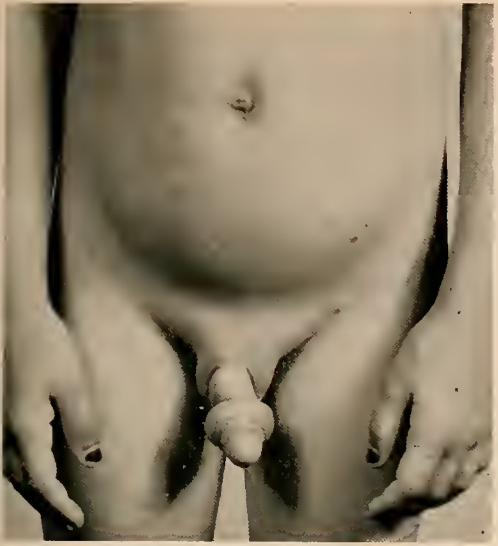

Fig. 3. Chigger bites on the abdomen. Edema of the prepuce produced by a chigger.

difficult to evaluate the usefulness of Robaxin in this case.

I know of a patient who responded well to intravenous Robaxin alone. I have used this drug in one case, but not until other treatment had been given. Since Robaxin has a low toxicity, it may prove useful in the treatment of black widow spider poisoning.

Chigger Bites

The chigger, or red bug, can cause more local misery for its size than any of the other insects discussed in this paper. Chiggers are the six-legged larvae of the trombiculid mites. When attached to man they inject a saliva which disintegrates cells. This produces inflammation and an allergic response in the host tissues. The resultant skin lesions are papules or vesicles closely resembling those of chicken pox. The chigger is sometimes found in the center of the lesion.

Impetigo, cellulitis, adenitis, or even bacteremia due to secondary infection may develop. When the chigger attaches itself to the foreskin, marked edema results. (fig. 3). Sometimes the victim has difficulty in voiding. Since pain and itching are unusual in this location and the chigger is embedded in the edematous tissue, the diagnosis of chigger infestation may be difficult to establish. The presence of chiggers on the child or a history of exposure will make the diagnosis obvious.

The application of finger nail polish, kerosene, collodion or Kwell (1 gamma benzene hexachloride in hydrophilic cream) may kill the chigger, but does little to relieve discomfort. Calamine lotion, Caligesic Ointment, oatmeal, or starch baths are used. To prevent scratching, the finger nails are cut short, and sedation is given at night. Antihistamines given by mouth reduce itching. Antibiotics are rarely necessary.

Since the discomfort is often severe despite treatment, the greatest service that can be rendered is to protect the child from exposure, by teaching him to avoid places where the mites are known to be plentiful, and by the use of repellents. *Off*, an aerosol spray which contains diethyltoluamide, is effective. This spray would not be applied in a close room, since one case of encephalopathy is reported to have followed its repeated use in a child's bedroom[7].

Since the same patients return each summer with severe reactions to chigger bites, efforts are being made to prepare desensitization material. Dr. W. C. Davison[9] informed me that several years ago Dr. Douglas Sprunt and Dr. Frank Swett performed some experiments on sensitivity to chiggers. They found some of the human volunteers very sensitive to the intradermal injection of ground-up chiggers. Their work was not completed. I have tried to duplicate the experiment, but it has been most difficult to collect or culture enough chiggers to prepare satisfactory skin-testing material.

Summary

Attention has been directed to four syndromes produced by insects. The puss caterpillar is becoming more common in this area. The early diagnosis of tick paralysis may be life-saving. A specific treatment for arachnidism is available. The chigger will continue to cause much misery until an effective method of treatment or desensitization is found.

A complete list of references will be included in the author's reprints.

Pediatrics Group Practice in a Prepaid Setting

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The social aspect of medicine has become increasingly important in recent years. Progress in preventive and curative medicine has been rapid since World War II, but concurrently increasing costs have presented more and more of a problem. A number of experiments have been tried to bring these two factors into some kind of balance. The present paper describes the practice of pediatrics under one of these new approaches to prepaid medical care. The organization to be described will illustrate just what can be accomplished when the professional and the consumer sit down with a mutual appreciation of each other's point of view.

Organization and Operation

Group Health Cooperative of Puget Sound was founded 15 years ago on the principles of group practice, prepayment for comprehensive coverage, consumer sponsorship, and joint planning for a better health-care program. The Cooperative is owned by the family members, who purchase one share each upon joining the organization, elect their own board of directors, contract with the autonomous medical staff for the provision of services, and prepay for these services through a monthly dues structure governed by themselves. This somewhat improbable organization now serves some 63,000 persons in the Seattle area and has just built a new 180-bed hospital entirely with its own funds. It operates a central major diagnostic and treatment clinic as well as two outlying general practice units. There are 61 members of the medical staff, representing all the major specialties in depth.

So long as the family pays its monthly dues, all members may receive unlimited office and home care, including all prescribed medicines and drugs (with the exception of maintenance vitamins), immunizations, x-rays, laboratory procedures—in short everything that is needed for comprehensive care. Hospitalization is covered up to 180 days for any single illness. Surgery, operating room, anesthesia, casts, special nurses when ordered, and the administration of transfusions (but not the blood) are all taken care of by the prepaid dues.

The medical staff is a separate unincorporated association all of whose members are on a full-time basis. This association contracts with the cooperative to provide the medical services described. Compensation is handled by means of a lump-sum budget which is distributed to its members by the staff. The ratio of physicians to subscribers is defined in the contract. The contract also provides time and expense allowance for postgraduate study, a month's vacation annually, sick leave, life and disability insurance, social security, and a retirement program.

The by-laws establish the structure of the medical staff organization and assign responsibilities to the appropriate committees and sections. Individual salaries are determined by the staff on the basis of training, qualifications, and meritorious performance. The net income approximates the average in the community.

The Department of Pediatrics

The Pediatrics Department consists of seven board-certified pediatricians and one in his required practice years. The pediatricians have their own departmental organization, arrange their own schedules, plan their own educational program. The section meets weekly for pediatric rounds in Group Health Hospital and attends teaching and demonstration rounds in the University and Children's Orthopedic Hospitals. General staff rounds, clinical pathologic conferences, and hospital staff meetings are also attended.

Each month the Pediatric and Obstetric sections meet jointly to review all maternal and newborn mortality and morbidity reports. This close working relationship between the two departments has contributed greatly to a very favorable neonatal mortality figure, averaging 12.8 deaths per thousand live births over the past five years.

The control of the newborn service is assigned to one pediatrician for a month at a time. This centralized responsibility, with decreased traffic through the nursery, has contributed to a very low incidence of infection.

The pediatric staff organization also provides that night and weekend duties are rotated throughout the department so that each physician has time for study, family life, relaxation or participation in community affairs. He is enabled to retain his competence, sanity, blood pressure, and position as *pater-familias* without the grinding round-the-clock interruptions of a busy solo practice.

Operation

In day to day operation the Cooperative is responsible for all supplies, personnel and facilities necessary to the practice of good medicine. All monies are collected by the central office, including private patient fees, and the appropriate records kept. Administrative, housekeeping, and economic matters are handled entirely by lay persons. The physician is therefore free to practice medicine as he thinks it should be practiced, without having to inquire whether the patient will be able to pay.

The annual budget of the organization is now over four million dollars—a sum quite enough to provide good, sound, intelligent care. The physician is conscious of cost only to the extent that he feels it a part of his duty to the staff and to the patients to use the available funds most intelligently. He works closely with the consumer on a variety of committees both in planning and conducting a medical care program under which producer and consumer are constantly pressing for quality and comprehensiveness. This close working relationship with the general public is relatively new to the experience of most physicians. It can be categorically stated that there has never been any attempt on the part of the lay members to interfere with the doctor-patient relationship or to attempt to dictate on matters pertaining to the practice of high quality medicine.

For the pediatrician working in such a setting this means that where diagnostic studies are needed they may be obtained. Where consultation is indicated, the entire medical staff is at his disposal. If advice is needed in an area not covered by the staff, the cost of an outside referral is covered by the budget. If one pediatrician would like the opinion of another, he has a colleague next door. No one need be tempted to perform procedures for which he is not specifically trained. It is a tenet of the staff, well observed, that there shall be no reluctance to ask for help in the interest of better care for the patient. With no premium on mass production for the physician, there is no point in trying to corner the market.

In the outpatient clinic the staff has developed joint teaching and consultation programs with the orthopedists and the dermatologists. In addition, young general practitioners who come to the staff with what is felt to be an inadequate background in pediatrics are given varying periods of training in the Pediatrics Department. Through these measures the pediatrician's own capabilities have been developed and his specialized knowledge passed on to the generalists. One tangible result: the oldest congenital hip dislocation referred to the orthopedic section for care in the past seven years has been five months.

Results

What is the response to such a program on the part of the patients? Experience indicates that Group Health Cooperative members present themselves faithfully for well baby care, immunizations, school, camp and periodic examinations, and show no reluctance to coming early in the course of an illness. To the extent that these habits challenge the staff to provide high quality care, there is surely the opportunity to do so. Furthermore, there has been very little abuse of the service, and that easily controlled. Abuse of service is regarded in the Group Health Cooperative as a symptom in itself. It can frequently be used as a index of the psychologic instability of the home.

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5,000	5,000 to 10,000	50.00 Weekly	$ 78.00	$ 39.50	$104.00	$ 52.50
5,000	7,500 to 15,000	75.00 Weekly	114.00	57.50	152.00	76.50
5,000	10,000 to 20,000	100.00 Weekly	150.00	75.50	200.00	100.50
5,000	12,500 to 25,000	125.00 Weekly	186.00	93.50	248.00	124.50
5,000	15,000 to 30,000	150.00 Weekly	222.00	111.50	296.00	148.50

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- The official Blue Shield emblem symbolizes national approval. For the Association, it also represents a cherished asset, one that has been built through untiring efforts, promotional expenditures—and with the understanding and cooperation of the Society itself for the past fifteen years.
- Through its extensive statewide sales organization, the Association continues to make Blue Shield available to North Carolinians. It is proud of its 1960 increase of over 61,000 participants—a gain of 12.78% over 1959.

In 1940, at the request of Dr. I. H. Manning, the House of Delegates permitted Hospital Saving Association to write Surgical and Medical programs. Thus, from the beginning, the Association has operated under the aegis of Medical Society leadership.

In 1946, Hospital Saving was included in the first group of Plans that were given the privilege of using the copyrighted Blue Shield emblem. National approval has been continuous; Hospital Saving has always considered it a sacred trust to protect and build the fine national and local concepts of Blue Shield.

More recently, the Medical Society instructed the Association to provide the DOCTORS PROGRAM of service benefits, which has grown in public acceptance to the point where over 31% of the Association's membership—and over 36% of its professional claims—are now under this program.

®

Investigation will often uncover a situation of stress which can be dealt with before it becomes a major crisis.

One final special feature of the Group Health program should be noted. The Co-operative puts out a news bulletin ten times a year, and additional news letters as needed. The material prepared by the Pediatric Staff concerns safety programs, immunizations, epidemics, and seasonal health reminders. Group membership meetings are also held in the evenings at which many topics of mutual interest have been explored. The staff has found that the information thus provided has contributed to a desirable sophistication on the part of the member parents.

The Group Health approach is but one of many probings in the field of medical care. In the current period of great social and economic change, it offers one possible method by which high quality medical care may be made readily available to the average American family at a cost which it is able and willing to pay. The program is essentially a partnership between physician and patient. For the physician it offers an unusual opportunity for the application of leadership in a climate of mutual interest and trust.

Sporadic Vitamin D Resistant Rickets

Report of a Case

Robert B. Tudor, M.D.
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Vitamin D resistant rickets was first recognized by Albright, Butler, and Bloomberg[1]. They believed that it represented an intrinsic resistance to the primary effect of vitamin D, and that deficient absorption of calcium from the gastrointestinal tract resulted in a low serum calcium content, causing parathyroid hyperplasia, hyperphosphaturia, and a decrease of the serum phosphorus. This disease may represent a congenital defect in the renal transport mechanism for phosphorus[2]. The lack of reabsorption of phosphorus probably keeps it at a permanently low level.

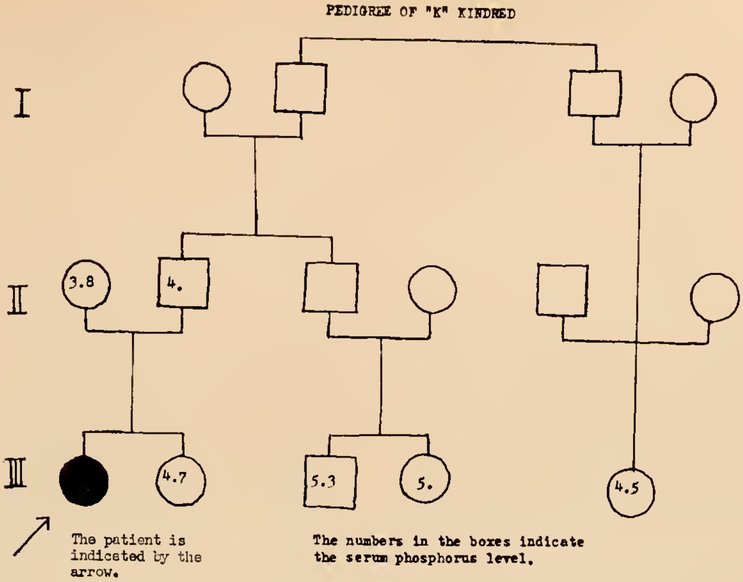
Genetic studies by Winters and others of several kindreds having vitamin resistant rickets of the "simple" type have demonstrated that:

1. Hypophosphatemia is superior to bone disease as an indicator of the presence of the abnormal gene.
2. Hypophosphatemia behaves in these families as a sex-linked dominant trait.
3. Hypophosphatemic females tend to have minimal, if any, skeletal disease, though there may be exceptions.
4. Hypophosphatemic males nearly always have moderate or severe skeletal disease.

Because this trait is carried on the X chromosome, an affected father cannot transmit the condition to his sons, but all his daughters will be affected. Vitamin D may raise the phosphorus level by increasing the reabsorption of calcium and decreasing parathyroid activity, or by directly affecting the renal tubules[2]. A case of sporadic hypophosphatemia has recently been reported in a female child with severe rickets. All her key relatives were normal. Aside from bone disease more severe than that seen in females with the sex-linked disorder, and the different hereditary background, no differences were found which would distinguish this case from the sex-linked variety of the disease.

It is the purpose of this paper to present another case of "sporadic" vitamin D resistant rickets. Three generations of this kindred were surveyed. No cases of rickets or hypophosphatemia were found in several key members of this family selected for study.

From the Department of Pediatrics, Quain and Ramstead Clinic, Bismarck, North Dakota.


PEDIGREE OF "K" KINDRED

The patient is indicated by the arrow.

The numbers in the boxes indicate the serum phosphorus level.

Figure 1

Case Report

The patient was born at term on January 28, 1954. Bowed legs were noted when she began walking at the age of 14 months. Roentgenograms taken on May 16, 1956, at the age of 28 months, disclosed bowing of the femurs, trumpeting of the distal portions, and widening of the epiphyseal plates.

On February 22, 1957, she was referred to this clinic for general examination. Marked bowing of the legs, beading of the ribs, and bone pain were noted. Her height was 34 inches (normal, 36.7) and her weight was 26 pounds (normal, 29.8). No cystine crystals were found in a bone marrow preparation, nor in the corneas by slit-lamp examination. The serum calcium was 11 mg. per 100 ml., the phosphorus 2.75 mg., and the urea 26 mg. Vitamin D was started in a dosage of 200,000 units daily. No calcium salts were given. The serum phosphorus was now 3.8 mg. per 100 ml.

On July 9 roentgen studies showed considerable improvement. The serum calcium was now 18.6 mg. and the serum phophorus 5 mg. per 100 ml. During the remainder of 1957 the serum phosphorus level varied from 2.9 to 3.0 mg. per 100 ml. The daily dosage of vitamin D. was reduced to 50,000 units when the serum phosphorus increased to 5 mg. per 100 ml. In November, 1957, renewal of rachitic activity made it necessary to increase the dosage, which was finally stabilized at 100,000 units daily. In April, 1958, when the patient was 4 years, 3 months of age, no rachitic activity could be detected by x-ray. She was now 35 inches tall and weighed 30 pounds. Vitamin D was temporarily discontinued, as the urea was then 60 mg. per 100 ml., but it was resumed on May 2, when the urea was recorded as 26 mg. per 100 ml.

Throughout the remainder of 1958 the calcium and urea values remainded normal, and the phosphorus values ranged from 3.3 to 3.6 mg. per 100 ml. The vitamin D dosage was reduced to 50,000 units daily until November 9, 1959, when anorexia and bone pain were noted.

At this time the patient's height was 41 inches and her weight 42 pounds. The blood urea nitrogen was 27 mg. per 100 ml., the serum calcium 11 mg., and the phosphorus 3 mg. Roentgenograms disclosed reactivation of the rickets at the knee and ankles. The dosage of vitamin D was increased to 100,000 units daily. By December, 1959, all symptoms had disappeared and her blood

Table 1

Chemical and Clinical Data on Normal Members ("K" Kindred)

Generation	Year of Birth	Age When Studied (years/months)	Sex	Height (inches)	Serum Phosphorus (mg./100 ml.)
II	1921	39	F	62	3.8
II	1923	37/8	M	70	4.0
III	1950	11/5	F	55	4.7
III	1951	9/10	M	51	5.3
III	1949	12	F	51.2	5.0
III	1950	10/7	F	50.5	4.5

chemistry values were within normal limits except for the low serum phosphorus (3.4 mg. per 100 ml.). The phosphorus rose to 4.9 mg. in January, 1960, but fell to 4.0 mg. in March. Roentgenograms taken in March, 1960 again showed improvement. The blood urea values during 1960 were within normal limits.

In November, 1960, her height was 43.2 inches and her weight 45 pounds. On March 24, 1961, the vitamin D was discontinued because of an elevated urea level (46 mg. per 100 ml.). The hemoglobin had fallen to 9.3 Gm., but the blood was otherwise normal. Iron was prescribed in a daily dosage of 100 mg. On April 19 the urea had risen to 60 mg. per 100 ml. and the hemoglobin to 12.1 Gm. The patient was asymptomatic and roentgenograms showed no rachitic activity.

Summary

A case of sporadic low phosphorus, vitamin D resistant rickets is reported. Though her relatives have no clinical or biochemical disorders, this patient does not differ from patients with the common "simple," sex-linked form of the disease.

Treatment of this condition consists of vitamin D in a daily dosage of approximately 100,000 units. The rickets will usually disappear under this treatment, though the serum phosphorus may remain low. The complications of vitamin D therapy, hypercalcemia, and uremia should be sought carefully, and the vitamin D should be discontinued if they occur.

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The secret of the care of the patient, lies in caring for the patient. Can we only care for the patient on the basis of an individual fee for service? We know that this is not so. The history and traditions of the voluntary hospital of the past belie it. Therefore, however desirable, or whatever advantages there may be in an economic structure of medicine based upon fee for service, let us not make it the essence of patient care, for we know that it is not.—Dunphy, J. E. Chirurgiam Virumque. Ann. Surg. 154: 4 (July) 1961.

Infantile Spasms and Hypsarhythmia

A Case Report

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Montgomery, Alabama

The syndrome of infantile spasms and hypsarhythmia was first described in 1954, by Gibbs, Fleming, and Gibbs[1]. They consist of very brief seizures in the form of head-nodding, myoclonic jerks, quivering, or "salaam seizures," in which the arms and legs are suddenly extended or flexed with concomitant dropping of the head. The salaam seizure may last less than one second and usually occurs in clusters lasting from one to fifteen minutes. Seizures are most common on awakening or during periods of drowsiness.

The characteristic electroencelphalographic findings are random slow wave and spike discharges of high voltage which change rapidly in duration and location—"multiple foci." These findings are present in sleeping and waking tracings[2].

The onset of this syndrome is usually between the ages of 3 and 12 months. Cases have been reported, however, in infants one day old, while in other patients the onset has occurred as late as 3 years of age[2].

Etiology and Prognosis

The etiology of the syndrome is obscure. Birth injury or brain damage, congenital defects of the central nervous system, and metabolic aberrations have all been implicated. Despite this list of possible causes, the etiology of the majority is "unknown."

The combination of infantile spasms and hypsarhythmia carries an extremely grave prognosis, since 87 per cent of these children become mentally retarded and 11 per cent die before the age of 3 years[1]. (In patients having infantile spasms without the electroencephalographic picture of hypsarhythmia, only 45 per cent are physically or mentally retarded[3]. Evaluation of treatment in this group, especially in those seen after the age of 2 to 3 years, is difficult, since the typical spasms usually disappear by the age of 3 or 4 years. Approximately 50 per cent will develop *grand mal* or other seizures.

Treatment

As disturbing as the poor prognosis is the lack of response by these children to treatment with the usual anticonvulsant drugs. However, Baldwin and Renner* recently reported favorable results with the use of acetazolamide (Diamox).

The first treatment suggested by Gibbs was the use of a broad spectrum antibiotic (Aureomycin, Chloromycetin, and Teramycin) for a period of 10 to 20 days, which ". . . in some cases resulted in normalization of the EEG and in subsequent normal mental and physical development."[1, 4]

In 1957 and 1958 Sorel and Dusaucy-Bauloye presented evidence showing that ACTH was highly effective for treating this entity[2]. In 1958 Low[5] reported favorable results with the use of ACTH and again, in 1959, Stamps, Gibbs and others[2] reported 60 consecutive cases treated successfully with this drug. They concluded that "... ACTH therapy results in a dramatic improvement in at least 30 per cent of such cases."

One attempt at dietary control has met with no success[6].

The following report follows a case from birth, through the development of the disease, to failure of treatment with a short course of cortisone, excellent response to ACTH, relapse after discontinuing the drug, and finally to an apparent "cure" with prolonged ACTH therapy, all substantiated with electroencephalographic findings.

Case Report

Present illness

A white male infant, aged 6 3/4 months, was first admitted to Maxwell Air Force Base Hospital on March 16, 1960, because of drawing in

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From Maxwell Air Force Base Hospital, Montgomery, Alabama.

*In a manuscript distributed at the Spring Session of the Academy of Pediatrics, Washington, D. C.

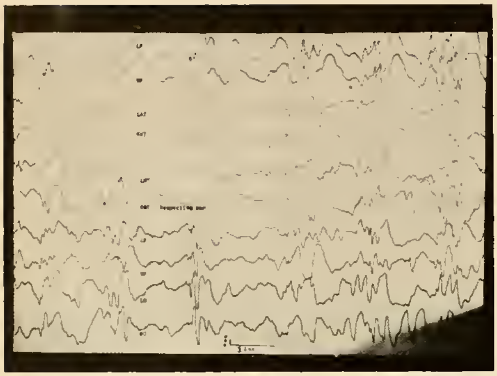

Fig. 1. EEG taken March 17, 1960.

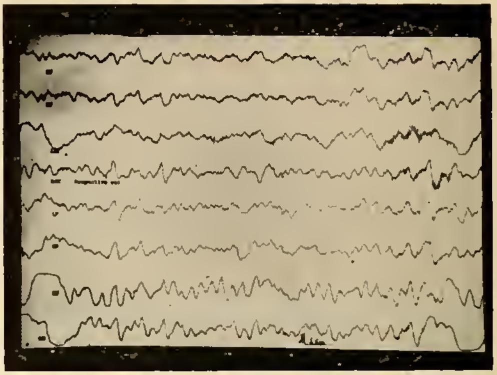

Fig. 2. EEG taken April 8, 1960.

his neck and arms." He had been considered well until approximately three weeks before, when his father first questioned his ability to hear. "Since then he just doesn't seem to pay any attention to anything." Five days before admission he began having episodes of flinging the arms outward or upward, dropping the head to the chest, and rolling up the eyes. The spells lasted but a fraction of a second. According to his mother, he had seemed "weak and sleepy" for the entire week before admission.

History

This child was born at term at Maxwell Air Force Base Hospital on August 23, 1959. The labor and delivery were extremely difficult. The onset of respiration was delayed 11 minutes, and breathing was not considered normal until 22 minutes after birth. His condition on admission to the nursery was poor. He was tube fed for four days because of a poor sucking reflex, but then he seemed to do surprisingly well. He was subseqently followed in the Well Baby Clinic, where his development was considered average. He smiled at six weeks, and turned from his abdomen to his back at 2¼ months. Just before admission he was sitting with support, and could sit alone for a few seconds.

Another child in the family, an 8 year old girl, had "mild" cerebral palsy and asthma. Her I.Q. was normal, and there was no history of seizures. The remainder of the family history was negative.

Clinical findings

The physical examination on admission was negative. There was no evidence of infection. The child could sit with support, and sit alone momentarily. Neurologic examination was also negative. There was good reciprocal motion in the arms and legs. No abnormal reflexes were elicited.

The hemoglobin, white blood cell count and differential were normal. Urinalysis, including tests for phenylketonuria and metachromatic granules, and examination of the cerebrospinal fluid were within normal limits. The only abnormal laboratory findings were the serial electroencephalograms.

Course of the disease

An EEG obtained the day after admission was typical of hypsarhythmia (fig. 1). The child was therefore started on oral cortisone in a dosage of 50 mg. per day. During the following five days he began having numerous showers of seizures, consisting of the salaam-type spasms and lasting 10 to 15 minutes. Because of the apparent rapid progression of the disease, ACTH was substituted for cortisone on March 22, in a dosage of 10 units (of the intramuscular gel preparation) daily. There have been no seizures since the first day of ACTH therapy, which was continued for a total of 14 days.

During this period the serial electroencephalograms done every three days showed marked improvement. On April 5, 1960, the tracing was slightly abnormal because of independent left and right temporal slowing." On April 8, the day of discharge, the EEG was within normal limits: ". . . The previously reported independent temporal slowing is not evident." (Fig. 2)

On follow-up examination one week later the left temporal slowing was again evident. The parents, however, were exceedingly pleased with the child's progress—"You'd never know he was the same baby." The following week he was reported to be much less active, and was sleeping much more, both day and night. At this time (April 22) the EEG showed considerable left temporal slowing and occasional independent spikes in the left temporal region, and a moderate amount of spike and wave activity (fig. 3). By April 29 the EEG was even more abnormal (fig. 4), and the child was "not paying attention to anything or anyone." There had been no recurrence of seizures.

The child was readmitted and ACTH treatment

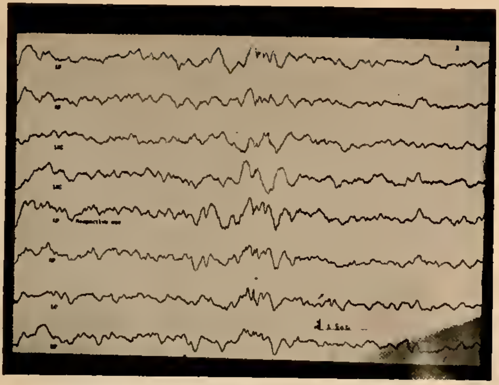

Fig. 3. EEG taken April 22, 1960.

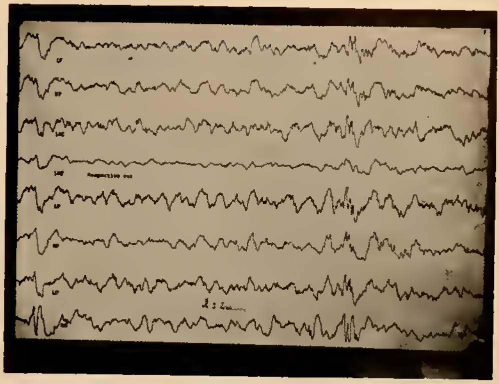

Fig. 4. EEG taken April 29, 1960.

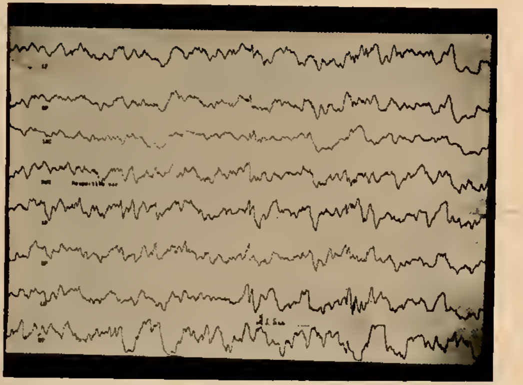

Fig. 5. EEG taken May 6, 1960.

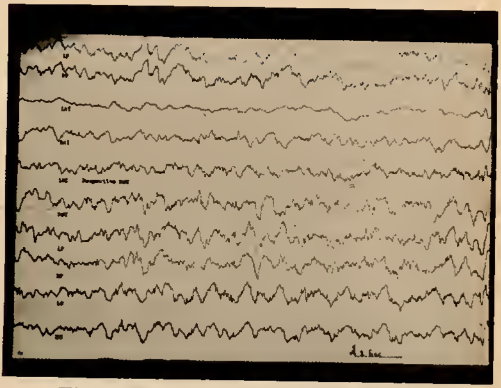

Fig. 6. EEG taken November 16, 1960.

was reinstituted in the former dosage. Laboratory tests were again within normal limits, except for the electroencephalograms. By May 7 the EEG was essentially normal (fig. 5), and the child was much more alert. He was discharged on a regimen of ACTH. He received daily doses for 30 days, then every other day for two weeks and finally every four days for two weeks. The last dose was given on June 30, 1960. Since then he has received no medication and his EEGs have remained normal.

Progress report

When seen on May 23, 1960, at the age of nine months the patient had begun to pull up and walk around his crib, wave "bye-bye," and was saying several words. At the age of 14 months he had been walking for 1½ months and had a vocabulary of eight words. At 16½ months he was well coordinated, had a vocabularly of 20 words, and was apparently well within the normal range of development. He was last seen at the age of 21 months, when the entire physical and neurologic examinations were negative. He is now saying phrases and an occasional sentence.

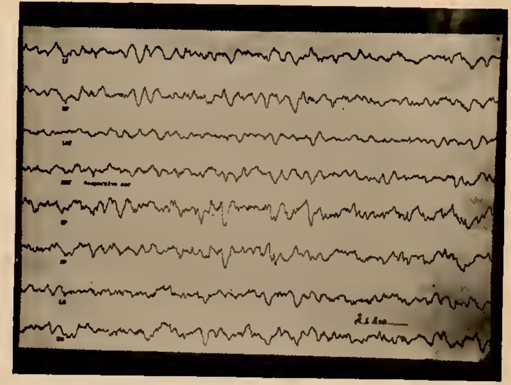

Fig. 7. EEG taken January 11, 1961.

Discussion

That the preceding report could represent a long series of coincidences cannot be denied. However, the clinical course, corroborated by the electroencephalograms, is against coincidence. The failure to respond to the short trial of cortisone followed by the dramatic response to ACTH cannot be explained on the basis of present knowledge.

In attempting to explain the rationale of ACTH therapy, Low[3] advanced five possibilities: (1) inflammation—not substantiated by pathologic studies; (2) hypoglycemia—blood sugar and glucose tolerance tests done to date have been normal; (3) allergy or autosensitization—a possibility; (4) demyelinating disease—but no other demyelinating disease has been known to respond to steroid therapy; and (5) the most intriguing—the possibility of a congenital or acquired enzyme defect or deficiency that might be altered by ACTH. This possibility has some support in the fact that in most series reporting the beneficial effect of ACTH in this syndrome, it is obvious that the sooner after the onset of the disease the therapy is started, the better the clinical results. The same is true for phenylketonuria and galactosemia—and it is noteworthy that this syndrome occurs in association with phenylketonuria.

Summary

A case of infantile spasms with hypsarhythmia is reported in which a short course of oral cortisone was unsuccessful. ACTH given intramuscularly gave dramatic results, followed by a "relapse" after cessation of treatment, and then most gratifying results after prolonged therapy with ACTH.

Acknowledgements

The author expresses appreciation to Lt. Col. George Cocks, USAF (MC), of the Department of Neurology, and to Mr. Tommy Estelle, Jr., of the electroencephalography laboratory, for their help in following this patient. Mr. Estelle is also responsible for the electroencephalograms reproduced in this paper.

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In assessment of the antihypertensive action of drugs in the clinic, one must always keep in mind the lability of the blood pressure and the ease with which nonspecific agents and placebos may appear to relieve symptoms and lower the blood pressure to some degree. The importance of the doctor-patient relationship, the powerful effect of a reassuring and enthusiastic approach to the patient, the effects of hospitalization, and other measures used to alleviate situational stresses necessitate the most critical approach, which unfortunately has not been followed in most published assays of antihypertensive drugs.— Grollman, A.: Clinical Pharmacology of Antihypertensive Agents, Clin. Pharmacol. & Therap. 1: 738 (Nov.-Dec.) 1960.

Viral Myocarditis In Infancy and Childhood

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Recent advances in virologic techniques —namely, the use of suckling mice and tissue culture—have resulted in clarification of some of the etiologic problems of myocarditis in infants and children.

Patients with myocarditis in this age group may be subdivided into two broad categories. Included in one group are those individuals who are infected with a viral agent that is largely cardiotropic. Predominant signs and symptoms of disease are therefore most often referable to the heart. Best known of this cardiotropic group of viral agents are the Coxsackie B viruses. The second group comprises those patients in whom organs and tissues other than the myocardium bear the brunt of the invading viral agent. A variety of diseases may involve the myocardium in varying degrees of severity; these include poliomyelitis[1], varicella[2], variola[3], measles[4], mumps[5], influenza[6], cytomegalic inclusion disease[7], psittacosis[8], epidemic hemorrhagic fever[9] and infectious mononucleosis[10]. The presenting and more obvious complaints are not usually referable to the heart, and the attention of the clinician under these circumstances is directed initially to other areas of the body.

The importance of Coxsackie viruses as etiologic agents in infant myocarditis was first recognized by Gear and Measrock in 1952[11]. These investigators studied an epidemic in 10 newborn infants who manifested symptoms of acute myocarditis or encephalomyocarditis shortly after birth. Six of these infants died, and inoculation of suckling mice with brain and heart suspensions from 2 of these cases produced lesions compatible with Coxsackie group B infection. Coxsackie type B3 was also isolated from the stool of a patient who recovered[12].

Since the original description and viral isolation in 1952, isolated cases[13] and small epidemics of encephalomyocarditis and myocarditis[14] in the newborn have been reported from various parts of the world. Well documented cases of myocarditis due to Coxsackie virus have been reported in older children and adults as well as in infants[15]. Strong support for the etiologic relationship of myocarditis to the Coxsackie B virus has evolved with additional isolation of these viral agents from the myocardium[12, 14a, 16], the brain[14a,c], the spinal cord[13b, 17], the blood[18], the kidney[19], and the liver[17,20] of patients dying with myocarditis.

Of the five serotypes of Coxsackie Group B viruses, only type 1 has not been implicated in the cases of myocarditis reported to date[13a, 17, 21].

Prognosis

The disease is usually much more severe in infants than in older children or adults. In the newborn baby myocarditis is not infrequently accompanied by other severe organopathy which may contribute to the high mortality rate seen in this age group.

Kibrick[13a], in a review of 45 neonatal cases of myocarditis due to Coxsackie B infection, noted that death occurred in 33. Most of the patients expired within one week after the onset of the illness, and 20 died within a day after they became clinically ill[13a]. It must be pointed out, however, that this death rate of 75 per cent is probably inordinately high, since most of the diagnoses were based on postmortem viral isolations.

Signs and Symptoms

The clinical picture in the typical severe case is one of abrupt onset, with nonspecific signs such as fever, listlessness or difficulty in feeding (table 1). Respiratory distress and cyanosis are noted in 4 out of 5 patients, and cardiac signs such as tachycardia, cardiomegaly, murmurs, and gallop rhythm are present in almost the same proportion. Less common symptoms are hepatomegaly

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*Recipient of the Lederle Medical Faculty Award.

Table 1
Symptomatology in Cases of Coxsackie Group B Infection Characterized by Myocarditis in the Neonatal Period*
(33 Fatal and 12 Non-fatal Cases)

Symptoms	No. Cases in Which Sign Was Present	No. Cases in Which Sign Was Sought	Per Cent
Feeding difficulty	31	37	84
Listlessness	29	36	81
Cardiac signs	30	37	81
Respiratory distress	24	32	75
Cyanosis	26	36	72
Fever	25	36	70
Red throat	7	11	64
Hepatosplenomegaly	19	36	53
Diphasic course	14	40	35
Central nervous system signs	11	41	27
Hemorrhage	5	40	13
Jaundice	5	40	13
Diarrhea	3	40	8

*From the data of Kibrick (13a).

(50 per cent), icterus (13 per cent), hemorrhagic diathesis (13 per cent), and diarrhea (8 per cent) [13a, 22]. Signs and symptoms referable to central nervous system involvement are present in almost one third of the patients, although pathologic changes within the central nervous system are noted in at least 3 out of 4 infants who died.

In about one third of the infants the disease followed a diphasic course. Nonspecific signs and symptoms of illness abated after 24 hours for a period of 1 to 7 days. This apparent recovery was followed by a more severe and often fatal attack characterized by fever, rapid respiration and pulse, cyanosis, signs of cardiac failure, and circulatory collapse[13a].

Pathology

Characteristic histopathologic changes consist of patchy interstitial edema, focal necrosis, and pleomorphic cellular infiltration with large mononuclear cells and histiocytes predominating[11]. Lesions are most pronounced in the heart, but are also present in the brain, spinal cord, liver, kidney and adrenal[13b,c, 17, 20]. These changes are strikingly similar to those observed in suckling mice experimentally infected with Coxsackie B virus[23].

Diagnosis

The diagnosis of myocarditis in infants is based on the presence of tachycardia, cardiomegaly and electrocardiographic abnormalities. The heart rate is usually over 200, and although the sounds may be of poor quality, no pathognomonic murmur is heard. A gallop rhythm is not uncommon and cardiac failure, if not present at the onset of symptoms, usually develops within a matter of hours. Inversion or flattening of the T waves are the most conspicuous electrocardiographic finding; depressed or elevated ST segments, inverted QRS complexes, prolonged P-R interval, or complete A-V dissociation may also be noted[16a, 21].

Differential diagnosis include the rarer entities such as glycogen-storage cardiac disease, anomalous left coronary artery, and medial necrosis of the coronary artery. A somewhat common and more difficult entity to differentiate from myocarditis is subendocardial fibroelastosis[24]. Ultimate differentiation may depend upon long-term follow-up in patients who survive the acute phase of the illness. Response to digitalis in myocarditis is often dramatic, with marked clinical improvement.

Digitalis in subendocardial fibroelastosis is only effective temporarily, for despite treatment, progressive enlargement of the heart results in intractable cardiac failure and death.

For definitive etiologic diagnosis, an attempt should be made to isolate the agent from the gastrointestinal tract of the patient in the acute stage of the illness or from the myocardium and other tissues at autopsy. Primary monkey cells in tissue culture as well as suckling mice may be used for these purposes, since both these hosts will support the growth of Coxsackie B viruses. Further confirmation of the diagnosis rests in a fourfold rise in titer of serum antibodies against the isolated viral agent. Ultimate identification as a Coxsackie B virus rests on appropriate serologic tests.

Epidemiologic Characteristics

Coxsackie B virus may be acquired by the infant either *in utero* or after birth. In 5 instances transplacental transmission of the virus appeared likely since in all cases the mothers had experienced minor illness compatible with Coxsackie B infection a few days prior to delivery. It is not likely that these infants were contaminated at the time of delivery, since the onset of illness was prior to the second day of life in 4 patients[13b, 17, 18, 25]. The fifth infant was delivered by caesarean section and became ill on the third day of life[13b].

Where investigations have been carefully pursued, most of the other reported cases of Coxsackie B encephalomyocarditis have been traced to direct postnatal contact either with a mother or with an attendant suffering from an illness which proved to be due to a Coxsackie B agent[13a, 25, 26].

It is of interest that the *epidemics* of myocarditis in newborn infants have been described by workers abroad, while only *isolated cases* have been documented by United States investigators. It might be postulated that the absence of epidemics is a consequence of a large-scale neonatal immunity —immunity acquired by transplacental transmission of antibodies from previously infected mothers. However, recent studies by Steigman and his co-workers[27] have shown that 40 to 75 per cent of mothers have no antibodies to Coxsackie B1, 3 or 5, and therefore—at least in his population group—a large percentage of infants are susceptible to Coxsackie B infection.

A more likely explanation for the absence of epidemics may be smaller nursery units and a shorter hospital stay for newborn babies in the United States. With the high infant mortality, the importance of protecting infants from contact with individuals who have possible Coxsackie infection is obvious.

Coxsackie B infections in older children and adults are usually benign, and may in fact be asymptomatic. Indeed most of the Coxsackie B infections in this age group result in pleurodynia, aseptic meningitis, or undifferentiated febrile illness[23].

Kagan and Bernkoph[15a], however, observed 7 cases of acute myocarditis in preschool and school children during an epidemic of upper respiratory infection. At autopsy Coxsackie virus was isolated from the myocardium.

It is apparent, however, that viral involvement of the myocardium is likely to be more frequent and more severe in the infant with Coxsackie B infection than in other age groups. This difference in relation to age is not easily explained.

Some corresponding observations in animals may be pertinent. It is well established that adult mice (except when pretreated with cortisone) are resistant to Coxsackie infection[28]. Suckling mice on the other hand are routinely used in the isolation of this agent. Newborn monkeys are similarly susceptible to Coxsackie B infection; the primary lesion in the newborn monkey is myocardial inflammation and necrosis, while injection of this agent into adult monkeys results in asymptomatic viremia[14c, 29].

The discovery of the role of Coxsackie virus in neonatal disease has led to etiologic solution of the puzzle presented by Fiedler's myocarditis. Many authors had postulated a viral etiology for this entity, and it seems likely that most cases previously described as Fiedler's myocarditis may in fact have been examples of Coxsackie infection[14a].

Other Viral Diseases

An intriguing question concerns the role of other viral diseases in the etiology and pathogenesis of chronic myocarditis. While chronic myocarditis is known to occur in children[21], there is no evidence so far that this syndrome bears any relationship to an attack of Coxsackie disease in early infancy[16a]. Long before viral agents could be isolated easily, investigators stressed the possible role of influenza, upper respiratory infection, poliomyelitis, and viral exanthematous diseases in nonvalvular cardiac disease[30]. Appreciation of myocardial involvement in these illnesses is directly proportional to the care exercised in detecting minimal abnormalities.

Jungeblutt and Edwards[16], in summarizing data on poliomyelitis, estimated severe involvement of the heart muscle in approximately 30 per cent of fatal cases. In another study of 27 fatal cases, microscopic evidence of myocarditis was demonstrated in every heart[31]. Electrocardiographic abnormalities may occur during the course of the acute illness in 32.4 per cent of the nonfatal cases[1a]. Of 33 fatal cases of influenza, acute myocardial involvement was present in 13[6b].

The ease with which minimal cardiac lesions may be overlooked in viral illnesses is emphasized by Hackel[2], who found focal interstitial inflammatory lesions in the myocardium of 7 patients who died with varicella. There were no cardiac symptoms at the time of death, and the original postmortem microscopic examination revealed cardiac abnormalities in only 1 case. More extensive sectioning revealed evidence of myocarditis in the other 6.

Whether direct viral invasion of the myocardium is the principal factor in the observed cardiac aberrations remains to be determined. Virus has been recovered from the myocardium in some cases of poliomyelitis[1b] and in 1 case of influenza[6b]. Direct invasion is certainly important in the pathogenesis of Coxsackie B myocarditis. In other situations it seems likely that a combination of factors contributes to cardiac pathology.

Viral invasion of the host produces systemic reactions that may result in profound metabolic, biochemical, immunologic and physiologic changes that may in themselves produce, predispose or aggravate myocardial lesions. Pearce and Lange[32] believe that cardiac anoxia is important in the pathogenesis of myocarditis accompanying viral diseases. Woodward and his associates[15c] suggest that an immunizing mechanism may be responsible for chronic myocarditis following repeated infections with viral agents.

New laboratory techniques may make it possible to elucidate the various factors involved in producing chronic myocarditis. Answers will be forthcoming only if careful clinical observations are combined with extensive virologic and immunologic studies over a prolonged period of time; to be meaningful these data must then be finally correlated with precise postmortem pathologic documentation.

Summary

1. Coxsackie B viruses produce severe encephalomyocarditis in infants and are responsible for cases of myocarditis in older children and adults.
2. Infants may be infected by transplacental transmission of the virus or by direct contact with infected mothers or attendants.
3. Epidemics in nurseries have been reported from abroad; isolated cases rather than epidemics are described in the United States.
4. Myocarditis in varying incidence and severity is known to occur in almost all diseases of known or suspected viral etiology.
5. The role of any of these agents in the production of chronic myocarditis remains to be elucidated.

A complete list of references will be included in the author's reprints.

Asthma In Children: Psychogenic Aspects

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Asthma is a disease with multiple etiologic factors, the most important varying from one person to another and from one time to another in the same person. These patients exhibit a propensity for reacting with bronchial contraction to stimuli that either do not affect the non-asthmatic person, or do so only when unusually intense. These stimuli include allergens, infections, emotional stress, noxious fumes, many drugs, local obstructions, physical changes in the environment (for example, changes in temperature and humidity), and impairment of cardiac function. It is also possible that two or more stimuli must be present in order to produce wheezing.

The allergic, infectious, and emotional factors seem to be most important in the disease. This discussion will focus on the role played by the emotions resulting from disturbed family relationships.

Evidence of the Importance of Emotional Factors

In recent years a great deal of attention has been given to the role of allergy in the etiology of asthma[1-8]. The gratifying success that usually results from treatment by hyperimmunization and avoidance of allergens is evidence of their importance. Several observations make it apparent, however, that other factors deserve attention.

One such observation is that in all reported series there is a residue of patients who do not exhibit improvement in spite of vigorous treatment directed against the allergens[5 9-12]. Another is the study reported by Long and his associates[13], who hospitalized 18 children with intractable asthma, most of them in an acute episode. As usually happens, all responded to hospitalization by rapidly becoming symptom-free. The house dust for each child was then collected by vacuum and circulated by a spray fan through his hospital room in an even stronger concentration than was present in his home. This was done for as long as 12 hours. None of the children had any reaction whatever. This study demonstrated that there was some difference between hospital and home other than, or in addition to, inhalants associated with the wheezing.

The consistency with which the asthmatic child exhibits marked symptomatic improvement after separation from the parents is one of the most significant pieces of evidence implicating disturbed family relationships[8 14-20]. As early as 1930 Peshkin and Murray[18] reported their experience with 41 children having chronic intractable asthma. Twenty-five were removed from their families to homes in the country; 16 remained with their families. None of those who stayed at home showed any improvement, while of those who were separated for an average of five years, 92 per cent were described as greatly improved or totally symptom-free. The changes were such that 70 per cent were able to maintain their improvement on returning home.

The most definitive reports have come from the Jewish National Home for Asthmatic Children in Denver, Colorado[16 20]. Admission is limited to children who have had intractable asthma for at least a year. They are admitted for a period of two years, and parental visiting is markedly restricted. The effect of climate was ruled out by admitting several children from Denver and nearby areas. In 1957 Tuft[20] reported experience with 500 patients at this home. Fifty per cent became free of wheezing within the first few days of admission; 30 per cent improved over the first year and were free of wheezing during the second year; 10 per cent stopped wheezing on admission but had recurrences during the pollen season; and 10 per cent were not improved.

Other studies suggest that asthmatic children and their parents have specific personality characteristics as compared with controls[7, 17, 21-25]. A criticism of most of these studies is that the examiner knew in advance which children were asthmatic and

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Table 1
Comparison of Allergic and Non-Allergic Children with Regard to Hostility

Mode of Expression	Per Cent Allergic	Per Cent Non-Allergic
Direct	20	83
Indirect	45	100
Displaced	63	100
Blocked	92	17

which were not. Another is that these personality characteristics may be the result of asthma rather than its cause. If, however, the personality disturbances are in some measure responsible for the precipitation of symptoms, whether they are primary or secondary is relatively unimportant as regards therapy; with regard to prevention the question is, of course, quite important. Another unanswered question is whether these findings are specific for asthmatic children or whether they are true of a child with any pulmonary disease.

Characteristics of the Asthmatic Child

Asthmatic children are described as submissive, fearful, lacking in self-confidence, passive, and compliant. All these terms are characteristic of a child who has difficulty in expressing anger and aggression directly. They are also immature, demanding, and clinging. This behavior suggests a desire for, and a fear of losing an extremely close, dependent relationship.

Miller and Baruch[23], in one of a series of studies of asthmatic children and their parents, observed evidence of hostility in 90 allergic and in 53 non-allergic children. They defined direct hostility as hitting, yelling, name-calling, or tantrums; indirect hostility as whining, untidiness, refusing to eat, and enuresis or forgetting; displaced hostility as that directed toward animals, another child, or property; and blocking as failure to express hostility in any way. Data were obtained from direct observation, reports of parents, and school reports. Their results are shown in table 1. The importance of this inability to express hostile impulses directly is confirmed by the observation that when during the course of psychotherapy the child becomes able to express anger more openly, the asthmatic symptoms improve[16, 20, 23].

The desire for dependency finds expression in several ways, the most obvious being overt behavior. The child often has the parent do for him what he could do for himself; he is reluctant to leave the company of the parent; and characteristically he wishes to be physically close to the mother —to be held in her arms. The wish may also be expressed symbolically. Long and others[13] analyzed a series of psychologic tests for claustral references—that is, to places that are small, dark, secluded, safe, warm, private, and concealing. A desire to be in such places is thought to indicate a wish to return to the prenatal state. Asthmatic children expressed such wishes three times as frequently as did non-asthmatic controls.

Parental Characteristics

The mothers of asthmatic children are invariably described as rejecting, this attitude being expressed both verbally and in their actions[8, 12, 16, 17, 21, 23, 24]. The parent of any child with a chronic illness inevitably has some feelings of rejection which are understandable. There is anger toward the child for being inferior and inadequate; resentment because of the expense, the sleepless nights, and the disturbance in the marital relationship; and guilt and loss of self-esteem because of having produced an unhealthy child. The rejection exhibited by the mother of the asthmatic child, however, is much more consistent, more overt, and more intense than that exhibited by mothers of children with what would seem to be comparable illnesses.

There is also considerable evidence that the rejection antedates the illness. Miller and Baruch compared the extent of maternal rejection in 201 allergic children and 110 children with other problems[23]. They defined the rejecting mother as "one whose behavior toward the child is such that she consciously or unconsciously has a desire to be free and considers it a burden, this rejection being expressed verbally by the mother." They found that 97 per cent of the mothers of asthmatic children were rejecting, while only 37 per cent of the mothers of non-allergic children displayed the same attitude. Studies of these mothers indicated that in childhood they had often felt deprived of maternal affection and experienced resentment because of it. As adults they seemed to be deprived and insecure individuals, with little capacity for the maternal role. Having to meet the demands of parenthood when they did not feel capable seemed, to these authors, to be the primary source of their rejection of the child.

The fathers of asthmatic children are conspicuous only by their absence. Their role in the family is passive; they may be retiring men who who are dominated by the mother, or, as is frequently the case, they are too preoccupied with their work to invest much time or energy in the family[16, 21].

Case Report

Bobby, a 6 year old white male, was seen in the pediatric allergy clinic of Duke Hospital for evaluation. His asthma had begun at the age of 8 months, at a time when his parents were beginning the separation which eventually led to divorce. He had only occasional mild symptoms until three months preceding this evaluation, when he, his mother, and his brother moved to North Carolina to live with her mother. Then Bobby began having severe wheezing almost daily. Psychiatric consultation was obtained at the suggestion of an intern who felt, on the basis of the mother's description of the episodes, that the child deliberately induced his wheezing.

Physical examination was unremarkable except for the chest, where there was slight hyperresonance and scattered wheezes and rhonchi. Chest roentgenograms revealed findings felt to be consistent with asthma. Skin tests for inhalants and foods showed a strongly positive reaction to peanuts, and a slightly positive reaction to egg and to dust.

The following excerpts are from interviews with the mother and with the patient. He was subsequently referred to the medical and psychiatric facilities in his local community and has been followed there.

Interview with the mother

How did you feel about having Bobby?

I didn't particularly want him at the time I had him, but actually I have more feeling for Bobby than I have for the older son. I mean, I guess I cling to him more or something. Now I don't know whether it's because he's sick . . . to be truthful, when Bobby was born he was an extremely ugly child . . . I mean, he just looked terrible. He just didn't look like he should've been mine. I had one beautiful child—Ralph, Junior [the older brother]. Ralph [the patient's father] and I weren't getting along too well when Bobby was born. Everything was just upset. And I had a hard time carrying him—a terrible time. And just different little things like that. And then when he was born and I saw him, he just didn't look he was mine. I never have believed that Bobby is mine, to be truthful. That sounds funny for a mother to say, but I just feel like he's not mine . . . He's a lot like his dad, but just not like me at all.

But now, whether—I tried to smother Bobby when he was born, as a matter of fact. I mean, I thought of it . . . But I breast-fed Bobby and I do love Bobby as much, I guess, or more than I do Ralph. I cling to him closer. He's not as affectionate as Ralph—there's a lot of difference in that.

In what way do you cling to him closer?

Well, when I go to bed I always hold Bob. I always sit and hold Bob at night watching TV. I'm always kissing him, and I pick him up on my back and piggy-ride him, and stuff like that that I never do with his brother. I mean, Ralph, he just doesn't require that . . . In others words, I have to take up more time with Bobby, because if I didn't, I just couldn't do anything with him . . . The little girl that had a baby that was in the bed next to me—the baby looked identically like my son Ralph. There was no difference whatsoever. And Bobby, he just didn't look like mine.

Do you mean that you have the feeling that the babies got switched?

I do, I always have. Because the other baby couldn't have looked absolutely like my first one, right on down to his little fingernails, and the way his little feet looked—everything—and then this one be so different . . . I wouldn't even name the child; that's how disturbed I was . . . As a matter of fact, I went into a state of shock.

Interview with the patient

Bobby was an average-looking 6 year old boy with a moderate speech impediment. He was well behaved, but was reluctant to talk.

What sort of fellow is your brother?

He tells me bad things to do and that's all I know.

What kind of bad things?

Like lighting matches, that's all he tells me.

Does your mother know that he told you about lighting matches?

No, she don't know about it. And if I tried to tell her, she'd go somewhere else.

You mean she wouldn't listen to you?

No; and she would to him. She would listen to what he says.

Does she treat you differently than she does him?

Yes.

What's the difference?

She treats him better than me. She likes him the most.

How much does she like you?

I don't know about that. I don't know nothing else.

Have you sometimes wondered whether she like you at all?

Uh-huh.

Do you get pretty mad at her sometimes because she doesn't seem to like you?

I just don't pay no attention to her: I don't know nothing else about nothing . . .

(At this point the child began wheezing audibly, this gradually subsiding when the conversation was turned to neutral subjects.)

Comment

This case is unusual by virtue of the severity of the disturbance in the relationship between the mother and child, but it well illustrates observations others have made of the asthmatic child and his mother. This mother made her rejection of the child quite clear, and her refusal to name the infant, together with her postpartum emotional disturbance, is objective evidence supporting the impression that her rejection antedated the onset of asthmatic symptoms. During the interview the child began wheezing (which was clearly audible on the tape recording) at the time that he seemed to be experiencing anger toward his mother because of her failure to love him. He was also attempting to assert his indifference to her.

The Conflict Between Dependence and Independence

As stated earlier, asthmatic children are immature and dependent. There are several possible explanations for a child's remaining overdependent. Quite often he has never been given an opportunity to be securely dependent during infancy, when total dependency is appropriate. Having been deprived of the vital assurance that he will be totally cared for, he does not develop the inner security that would permit him to move up from this position and assert himself as an independent human being. Instead, he clings desperately to what little security he has, and seeks in every way to achieve the state of total care that he should have experienced earlier.

A child may also remain immature because the mother implicitly demands it. She may need him to remain infantile so that she may retain complete control over him, or so he can be totally hers rather than a person in his own right. Such a mother, by her behavior, can make it clear to the child that if he wants to retain what love he has, he must remain immature. Jessner[17] has suggested that this, rather than the insecurity engendered by rejection, is the basis of the immaturity in the asthmatic child. Indeed, Abramson[24] has suggested that maternal rejection is secondary; that the mother needs and wants to keep the child dependent, and responds with anger and rejection when his self-assertiveness frustrates this desire.

Still another reason for continuing immaturity is that the child may have been taught to feel that progressing toward maturity is dangerous or impossible through a series of experiences in which he was "slapped down" for asserting himself, or by having been inculcated with the idea that he is incapable of becoming independent. He may also fear the sexual impulses that accompany maturation.

Although this close dependency is vitally necessary to the asthmatic child, it is invariably threatened. His acceptance is tenuous and he knows it. The inevitable anger toward the mother because of her rejection is frightening because he has learned that expressed anger is not tolerated and leads to further rejection.

The situation is further complicated by the frequent confusion of anger with self-assertiveness; indeed, the term aggression is used with both connotations. The child is often restricted and inhibited because he has come to feel that self-assertion in any form is the equivalent of angry defiance of the mother, and that it will lead to rejection. This feeling comes in direct conflict with the universal nature of a child, which is to become mature, independent, and self-assertive. Thus the asthmatic child inevitably finds himself divided between the urge to cling to the mother and the wish to be a separate and independent individual.

The asthmatic symptom probably occurs only when the conflict is actively present, and this explains why actual separation from the mother results in improvement. When the separation has been irrevocably established and the child has no choice but to be independent, the conflict dissolves. It is pertinent in this regard that when parents visit the children at the Jewish National Home for Asthmatic Children, wheezing recurs[16, 20]. The presence of the parent again makes dependency possible, and the conflict is reactivated.

Why this conflict is expressed in the symptom of asthma rather than in anxiety symptoms only—or in peptic ulcer, colitis, or some other illness—has not been determined. The familial occurrence suggests an inherited predisposition, and it is quite clear that there is great variation from one person to another, even in infancy, in the response to stress of different segments of the autonomic nervous system[25]. It is also possible that early trauma to the lungs produces structural alteration and increased susceptibility. It may be pertinent that the first act of the newborn child that establishes his physical separation from the mother is breathing; not breathing may thus be the physical expression of a desire to undo this separation and return to union with the mother. The asthmatic wheeze has also been described as a suppressed cry for help—suppressed because the child knows it will only bring the pain of disappointment, or perhaps because in another way he does not wish the dependency that the cry implies.

That conditioning might often be important was suggested by studies of adults by Dekker, Pelser, and Groen[26]. They had patients breathe into an apparatus into which were introduced specific allergens to provoke wheezing. After this had been done several times, the circumstances accompanying the induced attack were then sufficient to precipitate an attack without the allergen. The patients had attacks when inert substances were introduced, and some began having them as soon as they walked into the room. Thus if a child experienced particular feelings and impulses at the time of an asthmatic attack, perhaps caused by allergens or infection, subsequent attacks might be precipitated when the same feelings are aroused, no other stimulus being necessary.

These are possible mechanisms by which this emotional conflict might result in asthmatic wheezing. Which are, in fact, involved remains to be demonstrated.

Therapeutic Implications

Treatment of the asthmatic child should always begin with medical measures, which in most cases will suffice to control the symptom. Inhalants should be avoided by taking adequate precautions against dust. The value of hyperimmunization has recently been reaffirmed[11], and if the asthma is severe, this measure should be employed. Breathing exercises are often useful, and appropriate medication should be prescribed to alleviate the attacks. Controlling the wheezing usually results in an improved relationship between the child and his mother; her resentment of him because of his illness is diminished, and he has fewer occasions on which he has genuine reason to feel helpless.

During the course of medical management the physician can help improve relationships between members of the family, using his authority to oppose moves on the part of the parents to keep the child helpless, immature, and invalided. He can also attempt to teach both mother and child healthier ways of handling emotions than the suppressive and repressive techniques they have employed. Miller and Baruch[23] succinctly described how this can be accomplished, in some measure, during the one or two minutes involved in giving vaccine. The doctor may say something like this: "I'm going to give shots that will hurt. I know you will be angry with me and you may say 'ouch' or 'damn' if you want, but sit as still as you can." This statement exemplies a healthier way of handling negative feelings in that it (1) names the feeling of anger in a matter-of-fact manner; (2) identifies the person at whom the anger is directed; and (3) suggests acceptable channels through which the feeling can be expressed.

In a small percentage of cases psychotherapy is indicated. It would possibly be of some benefit in all instances, in view of the consistent pattern of disturbed family relationships; but this is obviously not practical in our present scheme of medical care. In the large majority of cases it is not necessary for control of symptoms. In general, psychiatric evaluation is indicated for those children who give evidence of emotional disturbance over and above the asthma, and in those with intractable asthma.

In children with severe intractable disease, separation from the parents may well be indicated. This might be accomplished by admission to an institution designed for long-term care of the asthmatic child, or by placement with relatives or in a foster home for a year or two. It is important to make it clear that the mother must not maintain close contact with the child, but this recommendation should be presented in terms of a change of home environment, in order to avoid arousing guilt on the part of the parents.

Summary

The asthmatic child is immature, seeks dependency, and is inhibited in his expression of anger and self-assertiveness. The mothers are characteristically rejecting, and there is some indication that this attitude precedes the onset of asthmatic symptoms. The fathers are usually passive in family relationships.

The psychodynamics that lead to asthmatic wheezing seem to involve a conflict between an urge to remain dependent and a need to be self-assertive and independent.

Medical management is the primary mode of therapy and will suffice to control symptoms in most instances. During the course of treatment the physician can intervene in favor of healthier family relationships and can encourage healthier ways of dealing with emotions. Psychotherapy may be necessary in some cases involving emotional problems in addition to the asthma, and where the asthma is intractable. In children with severe, long-standing, intractable asthma, prolonged separation from the parents is indicated, when feasible.

A complete list of references will be included in the author's reprints.

The Management of Neonatal Jaundice

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Neonatal jaundice can be one of the most difficult problems of management encountered by the physician. While the accumulation of large quantities of bilirubin in the blood stream is always dangerous, the serum level required to produce kernicterus (the most serious complication of neonatal jaundice) is variable. Until more is known about the factors which potentiate the toxic effects of unconjugated bilirubin, the management of neonatal jaundice must be based largely upon clinical experience.

Diagnosis of Jaundice

Since all infants are subject to the development of dangerous levels of serum bilirubin during the first week of life, a careful daily examination is important. If possible, the infant should be removed from the incubator or crib and examined for the presence of jaundice in direct sunlight by blanching the skin of the nose. (This maneuver tends to decrease the masking effect of the newborn's plethora.)

Mild, inconsequential icterus (physiologic jaundice) is said to develop in 30 to 80 per cent of normal newborn infants[1]. Physiologic jaundice of the newborn is a diagnosis of exclusion, however, and must be rejected in the presence of any of the following findings[2]:

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1. The onset of visible jaundice within 36 hours after birth (48 hours in the premature infant).
2. An indirect serum bilirubin greater than 15 mg. per 100 cc. (except in the premature infant).
3. Serum bilirubin rising at a rate greater than 5 mg. per 100 cc. in 24 hours.
4. Failure of the serum bilirubin to decline within 96 to 120 hours after birth.

Etiologic Diagnosis

Since jaundice is a symptom rather than a disease, effective therapy depends upon correct diagnosis of the underlying disease. An outline of the conditions which may produce jaundice in the newborn is given in table 1. The presence of some of these conditions may be anticipated during pregnancy, and in such cases the pediatrician and obstetrician should cooperate in making preparations to deal with any emergency that may arise after delivery.

In the case of iso-immune hemolytic disease of the newborn, particularly Rh erythroblastosis, a reasonably accurate prognosis concerning the severity of the disease can often be made from information regarding prior sensitization of the mother, neonatal jaundice in children born previously, the husband's genotype blood[3, 4], and serial anti-Rh antibody titers throughout pregnancy. Even when all information points to a good prognosis, however, the need for emergency treatment in the neonatal period sometimes arises.

When jaundice occurs unexpectedly in the newborn, it is important to obtain a careful family history regarding the presence of hemoglobinopathies, anemia, and jaundice. In the mother's history, the following points are relevant: possible exposure to toxins, the length of gestation, the complications of labor and delivery, recent viral infections such as hepatitis or herpes simplex, drugs taken during pregnancy (prescribed and proprietary), and anesthesia and drugs given during labor (especially vitamin K or one of the sulfonamides).

Table 1

The Causes of Neonatal Jaundice

I. Hemolytic disease of the newborn
*A. Rh incompatability
*B. ABO incompatibility
C. Lesser group incompatibility
II. Infectious diseases
A. Viral diseases
1. Cytomegalic inclusion disease
2. Neonatal hepatitis
3. Disseminated herpes simplex
B. Spirochetal diseases
1. Congenital syphilis
2. Leptospiral jaundice
C. Protozoan disease—toxoplasmosis
*D. Bacterial diseases
1. Intrauterine sepsis
2. Extrauterine sepsis
3. Other acquired infections
III. Heredofamilial diseases
A. Galactosemia
B. The Crigler-Najjar syndrome
C. Hereditary spherocytosis
D. The congenital nonspherocytic hemolytic anemias
E. The Dubin-Johnson syndrome
*F. Diabetes in the mother
G. The hemoglobinopathies
IV. Obstructions of the biliary tree
*A. Inspissated bile syndrome
*B Biliary atresia
V. Miscellaneous diseases
A. Cretinism
B. Mongolism
C. Pyloric stenosis
VI. Traumatic conditions
A. Subdural hematoma
B. Subarachnoid hemorrhage
C. Absorption of other large hematomas
VII. Factors potentiating the physiologic jaundice associated with transitory errors in the metabolism of the newborn
*A. Prematurity
*B. Anoxia
*C. Drugs
*D. Respiratory distress
*E. Dehydration
F. Miscellaneous

*Common

Laboratory tests

Blood from both the infant and the mother should be typed for the *ABO blood group* and the *Rh factor*. These tests, combined with the *direct Coombs' test* on the infant's blood, will reveal most blood group incompatibilities. Special immunologic tests for rare blood groups are occasionally useful.

A *blood culture* should be made in every case of unexplained neonatal jaundice. Cultures of blood obtained from the umbilical vein often represent the presence of bacteria on the umbilical stump rather than in the blood stream. Blood from the placental cord can be obtained for culture in cases where the possibility of sepsis is suspected because of early rupture of the membranes, prolonged labor, or infection in the mother. A large sample of placental cord blood for culture can easily be obtained at delivery, without any trauma to the newborn infant. This sample should be taken prior to delivery of the placenta, after the maternal end of the cord has been cleaned in an appropriate manner, and after a small amount of blood is allowed to flow through the cord in order to prevent contamination by the vaginal flora.

A *serologic test for syphilis* should be made on cord blood or on blood drawn from the mother at the time of admission. If toxoplasmosis is suspected, serum from the infant and mother can be sent to a suitable laboratory for a *toxoplasmosis dye test.*

A *hemogram* on cord blood or blood drawn from the infant is, of course, essential. Hemolytic disease of the newborn is often associated with severe anemia, and a low hemoglobin is an important early sign of impending difficulty. A reticulocyte count above 5 per cent suggests the possibility of hemolytic anemia. A total white blood cell count below 12,000 or above 25,000 (after correction for nucleated red blood cells) suggests the possibility of sepsis.

In the presence of an ABO group incompatibility, the finding of 2 or more spherocytes per high power field signifies that ABO erythroblastosis is likely. Spherocytes can also be found in the blood smears of newborn infants with hereditary spherocytosis, particularly those who develop a crisis shortly after birth. Although spherocytes are not increased in all types of hemolytic anemia, their presence in increased numbers is pathognomonic of a hemolytic process. The finding of more than 5 nucleated red blood cells per hundred white blood cells indicates either a hemolytic reaction or anoxia. Sepsis should be suspected if a relative agranulocytosis is found.

In some infants with an iso-immune hemolytic anemia, the typical findings in the reticulocyte count and blood smear may disappear within 48 to 72 hours after birth. This phenomenon presumably reflects an aplastic type of crisis.

Tests of the urine which may be helpful include a Benedict's test for reducing substance and a test tape for glucose; these can be used to rule out galactosemia in infants who become jaundiced after feedings have been started. In cases of cytomegalic inclusion disease, inclusion bodies may be found in a centrifuged sediment of fresh urine stained with methylene blue or Wright's (Wilson's) stain. If sepsis is suspected, the urine should be examined for bacteria and pyuria, and a urine culture should be made.

While *roentgen examinations* are rarely necessary, skull films sometimes demonstrate intracerebral calcifications which are suggestive of cytomegalic inclusion disease or toxoplasmosis. When the infant is having respiratory difficulty, a chest film should be made to rule out pneumonia, atelectasis, or hyaline membrane disease.

Subdural taps are rarely indicated except in occasional cases where jaundice follows a difficult, traumatic, or precipitous delivery. The symptoms of subdural hemorrhage may mimic kernicterus, and the absorption of blood from the subdural hematoma may lead to jaundice.

In cases of meningitis associated with neonatal sepsis, a rapid, tentative bacteriologic diagnosis can be obtained by *lumbar puncture,* with the demonstration of bacteria on smear or culture of the cerebrospinal fluid. Finding the crescent-shaped toxoplasmosis parasite in the cerebrospinal fluid confirms the diagnosis of neonatal toxoplasmosis. In interpreting the chemical determinations on the cerebrospinal fluid, one must keep in mind that hypoglycemia and elevation of the protein content of the cerebrospinal fluid are not abnormal in the newborn.

Xanthochromia and a few crenated red blood cells can be found in the cerebrospinal fluid of normal newborn infants. Large numbers of red cells, however, indicate subarachnoid hemorrhage. If bloody cerebrospinal fluid which clots is obtained on lumbar puncture, it is probable that a vessel has been punctured during the procedure, allowing venous blood to mix with the spinal fluid. In such cases the tap should be repeated immediately at a higher interspace, while the first needle is left in place.

To protect other infants in the same nursery, it is important to obtain *nose and throat cultures* from newborn infants suspected of having an infection. The respiratory passages, like the gastrointestinal tract, are normally sterile at birth.

Under some circumstances, *cultures of the stool and of amniotic fluid, placental membranes, umbilicus* and *other areas* may help in establishing the role of infection in cases of neonatal jaundice.

General Principles of Management

Early feedings of water and dextrose may tend to prevent dehydration and provide adequate carbohydrate. If oral feedings cannot be tolerated within 48 hours after birth, parenteral infusion should be considered.

Treatment with antibiotic agents is justified in any infant with unexplained pathologic jaundice. Until sepsis can be ruled out or the infecting organism can be identified, antibiotic therapy should cover both gram-positive and gram-negative organisms.

The neurologic status of the jaundiced infant should be evaluated several times each day. The signs of impending kernicterus appear most frequently between 48 and 120 hours after birth, and consist of diminished spontaneous movements, lethargy, failure to nurse, and alternating episodes of apnea and tachypnea. Since the onset of these signs is usually followed shortly by kernicterus[5], their appearance is an indication for immediate exchange transfusion.

Serial serum bilirubin determinations are necessary to determine the course of the jaundice. Since this laboratory procedure is subject to errors of 10 to 15 per cent[6], however, a borderline determination should never be used as the sole indication for exchange transfusion.

Blood for special immunologic studies, for the toxoplasmosis dye test, and so forth must be obtained prior to the first exchange transfusion.

Specific Therapy of Conditions Producing Jaundice

ABO erythroblastosis

Erythroblastosis due to ABO incompatibility rarely produces an anemia severe enough to require transfusion. An exchange transfusion is indicated if the serum bilirubin exceeds 20 mg. per 100 cc. A proper blood for transfusion in such cases is fresh, type O, Rh-positive blood of a low anti-A and anti-B titer, to which A and B substances have been added. The *ideal* blood is one made of cells which are compatible with the mother's serum (usually type O), resuspended in plasma taken from blood of the infant's major ABO type.

Rh erythroblastosis

If symptoms are present at birth, an immediate exchange transfusion is indicated. If the infant has no symptoms at birth, blood should be obtained from the cord for determinations of the hemoglobin, hematocrit, and bilirubin, as well as for a direct Coombs' test. If the hemoglobin is 13 Gm. or less, if the indirect serum bilirubin is greater than 6 mg. per 100 cc., or if there is a previous history of a severely affected infant, exchange transfusion should be undertaken immediately. In other cases, exchange transfusion can be delayed until signs of impending kernicterus develop, or until the indirect serum bilirubin reaches the level indicated below:

Age (Hours)	**Indirect Serum Bilirubin (Mg. per 100 cc.)**
0-24	10
24-48	14
48-72	17
72 plus	20

In some cases it is necessary to repeat the exchange transfusion. This should be done if the serum bilirubin nears a level of 20 mg. per 100 cc., unless the infant is near the age when a spontaneous fall in the bilirubin level can be expected (120 hours in the premature or 96 hours in the term infant). If any symptoms suggestive of kernicterus develop when the bilirubin level is greater than 15 mg. per 100 cc., the exchange transfusion should be repeated immediately.

Other conditions

Hereditary spherocytosis, the hereditary nonspherocytic anemias, cytomegalic inclusion disease, and *toxoplasmosis* are treated symptomatically, with exchange transfusions whenever the serum bilirubin exceeds 20 mg. per 100 cc. or kernicterus is considered imminent.

Physiologic jaundice of the premature is best managed by providing adequate hydration, either orally or intravenously, beginning within 48 hours after birth. Exchange transfusion should be performed if the serum indirect bilirubin exceeds 20 mg. per 100 cc., or if symptoms of kernicterus become apparent. Infants with respiratory distress are poor candidates for exchange transfusion; in such cases the procedure should be withheld, if possible, until the infant is in better condition. Since the incidence of kernicterus is probably less than 10 per cent with serum bilirubin levels below 30 mg. per 100 cc.[7], an expectant approach may be justified in the moderately jaundiced premature infant who has another life-threatening complication.

Exchange Transfusion

While a description of the procedure for exchange transfusion is beyond the scope of this article, a few points concerning its use should be made here.

Necessary precautions

In patients with borderline indications, the risks of kernicterus must be balanced against the risks of exchange transfusion. While sudden respiratory distress or symptoms related to the central nervous system may be signs of kernicterus, it is in infants with these symptoms that exchange transfusion has its highest mortality. The decision to undertake the procedure in such a situation must depend not only upon the clinical condition of the infant but also upon the experience of the physician and the available facilities. In some cases of severe hemolytic disease, a modified exchange transfusion may serve to adjust the venous pressure and improve the circulating red cell mass. This procedure has a definite place in the management of the pre-hydroptic infant who is in congestive failure at birth and is too ill to survive a total exchange transfusion.

The major cause of mortality associated with exchange transfusion is probably the undue exposure of the seriously ill newborn infant. No infant should be out of the incubator for more than two hours, and if respiratory embarrassment or other danger signals develop, he must be returned to the incubator immediately. If the infant is in very poor condition, it may be possible to perform an exchange transfusion without removing him from the Isolette.

Excessive cooling during an exchange transfusion should be minimized by setting the thermostat in the exchange room up to 80 F. and by allowing the blood used for transfusion to come to room temperature before it is given. (It should be gently shaken at frequent intervals to prevent sedimentation.) During the procedure, the infant should be kept at a stable temperature by external heat (hot water bottles wrapped in towels), if necessary. Under these circumstances, the body temperature seldom falls more than 0.5 to 1 C.

The blood to be used for the first exchange transfusion should be cross-matched with both the mother's and the infant's blood. If immediate exchange transfusion is anticipated, the cross-match using only the mother's blood should be done prior to delivery. Blood for repeat exchange transfusions should be cross-matched with the original blood samples (mother's and infant's), and a post-exchange sample of the infant's blood should be used to recheck the ABO group. Blood for subsequent exchange transfusions should be of the same major group that was used in the first procedure.

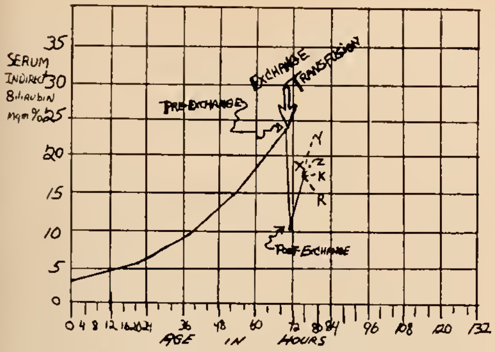

Fig. 1. Method for determining the need for a second exchange transfusion, regardless of the cause of neonatal jaundice. The predicted indirect bilirubin four hours after the exchange (x) is three-fourths of the pre-exchange level. The indication for repeat exchange is based on the rate of rise after the fourth hour (x-y, x-z, x-k, or x-r) and its relation to the height of the serum indirect bilirubin and the age of the patient.

Indications for repeat transfusion

An exchange transfusion of 70 to 80 cc. per pound of body weight can be expected to reduce the serum bilirubin level by 50 per cent. Bilirubin re-accumulates in the serum rapidly following exchange transfusion, and within four hours usually reaches a level approximately 75 per cent of the pre-exchange level. After this period of re-equilibration, the rate of increase slows, and an hourly rise of more than 1 mg. per 100 cc. is, generally speaking, an indication for a second exchange transfusion. This rate of increase can be calculated by plotting the expected rebound following an exchange transfusion and comparing it with a serum bilirubin determination made six to eight hours following the exchange (fig. 1). The condition and age of the patient, as well as the pre-exchange bilirubin level, must of course be taken into consideration in making the decision to perform a second exchange transfusion.

Summary

Management of the newborn infant with jaundice requires close observation, the use of well chosen diagnostic tests, and institution of therapeutic procedures. The physician can not depend solely on technical and laboratory aids, but must be able to appreciate the significance of the clinical signs and symptoms which occur in the jaundiced newborn. Adherence to unchangeable criteria as indications for exchange transfusion may lead to difficulty.

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An interesting and encouraging special study was carried out on people who had at one time been sufficiently overweight to merit substandard insurance and who later, by reason of weight reduction, were able to qualify for standard rates . . . The mortality experience among those who reduced their weight was comparable to the experience among those in the standard groups. These results are in accord with findings of an earlier insurance study and should offer to the clinician statistical justification and incentive for a weight reduction program.—Hutchinson, J. J.: Clinical Implications of an Extensive Actuarial Study of Build and Blood Pressure, Ann. Int. Med. 54: 94 (Jan.) 1961.

Phenylketonuria with Borderline and Normal Intelligence

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Phenylketonuria, formerly known as phenylpyruvic oligophrenia, is one of the metabolic disorders that lead to mental retardation for which a rational form of treatment is available. Its victims have poor utilization of phenylalanine due to a defect in the enzyme, phenylalanine hydroxylase. Phenylpyruvic acid and other related compounds are excreted in the urine in excessive amounts. There is an elevation of phenylalanine in the blood, which is thought to interfere with normal development and function of the central nervous system. The majority of patients will have severe mental retardation and, usually, convulsions. A secondary defect in the utilization of tyrosine results in poor pigmentation of the skin, hair and iris. Frequently, some degree of eczema may be present, and there may be a peculiar aroma (sometimes referred to as a mousy odor) about the patient and his urine.

A diagnosis can be made very simply by testing 5 or 10 cc. of urine with about 10 drops of 10 per cent ferric chloride. The urine should be made acid before testing and should be tested within a few hours after voiding. A heavy blue-green or grey-green color is diagnostic. Quite rarely a false-positive green color may be obtained when the urine contains certain other substances—namely, bile, catecholamines (pheochromocytoma), homogentisic acid (alcaptonuria), and substances present in maple syrup disease[1]. Detection of these substances is equally important, however, and contributes to the diagnostic work-up of the patient. In newborn babies, the appearance of phenylpyruvic acid in the urine is usually delayed, and the test should be repeated at a few weeks of age. Ferric chloride solution is readily available, but a simplified procedure using Phenistix (Ames) makes possible this determination on a wet diaper or a urine specimen voided at home.

The vast majority of patients with phenylketonuria have intelligence quotients of 30 or less. If treatment with a diet low in phenylalanine is begun within the first few months of life, damage to the central nervous system can probably be avoided. Treatment started from six months to two years of age provides diminishing returns. After 2 years of age there may be some improvement in behavior, the electroencephalogram, and pigmentation, but no increase in the I.Q. can be expected.

Although mental retardation is usually severe, about 2 per cent of all patients, with an apparently similar biochemical defect, will have borderline or normal intelligence. Of somewhat more than 1,000 cases of phenylketonuria reported in the literature, a total of 26 patients (including our cases) had I.Q.'s above 60. Knox[2] listed 20 patients reported to have borderline or normal intelligence. Sutherland and others[3] reported 2 patients, aged 4 and 7 years, with I.Q.'s of 93 and 67 respectively. Both of these patients had behavioral difficulties and showed decided improvement when placed on diets low in phenylalanine. These authors referred to a patient of Caudle whose I.Q. was 120 at 3½ years of age. Allen[4] reported following a child who had an I.Q. of 112.

Case Reports

Case 1

A 6 year old boy was first seen at the Jefferson Medical College Hospital on January 19, 1959, because of retarded development and hyperactive behavior. Past history revealed a normal pregnancy and delivery; the birth weight was 8 pounds, 15 ounces. He walked at 16 months of age, began using single words at 1 year, and put words together in short sentences at about 2½ years. He had an extremely short attention span and preferred to play by himself. Both parents are healthy and obviously of normal intelligence.

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There is only one sibling, a 9 year old girl, who will be described in case 2.

On physical examination, he appeared to be of normal stature and in good general health. He was blond, blue-eyed, and had some roughness of the skin. A definite musty odor was detectable, particularly about the scalp. He appeared somewhat dull mentally, but his speech was clear. The cranial nerves were intact, and motor function showed only a mild diffuse clumsiness. The sensory responses appeared to be intact, at least to touch and pain. The reflexes were all normal.

On January 22, 1959, his I.Q. was 71 as measured by the Stanford Binet test. Personality testing was not possible, but the Vineland Social Maturity Scale revealed a social quotient of 76, consistent with the I.Q. He was quite fearful, and emotional factors may have interfered to some extent with intellectual function.

In a routine study his urine gave a positive test for phenylpyruvic acid. The test was again positive when repeated on two occasions. On February 5, 1959, he was placed on a diet low in phenylalanine, and when tested again on May 22 the urine showed no qualitatively detectable phenylpyruvic acid. The parents reported a definite improvement in behavior following treatment. He was subsequently followed at St. Christopher's Hospital for Children in Philadelphia. The first serum phenylalanine determination, while he was on the diet and while the urine tests were negative, was 10.6 mg. per 100 ml. It has generally been maintained at less than 5 mg. per 100 ml. since that time. Later intelligence tests have shown little improvement in intellectual performance.

Case 2

The 9 year old sister of the patient in case 1 had a positive reaction when given the urine ferric chloride test in a follow-up of the family. Past history revealed toxemia of pregnancy from the seventh month of gestation until delivery. The delivery itself, however, was uneventful and the birth weight was 9 pounds. The patient walked at 11 months, began to use words at 12 months and short sentences before the age of 2 years. She has developed quite normally as far as the parents have observed, and has successfully completed the third grade in school. The ferric chloride test was negative in both parents.

On physical examination the child appeared to be perfectly healthy and in no distress. She also was blond, blue-eyed, and had a musty odor about the scalp. Neurologic examination was within normal limits.

On May 22, 1959, the WISC revealed a verbal score of 96, a non-verbal score of 94, and a full-scale I.Q. of 95. There was considerable scatter on the verbal tests, but not on the non-verbal. There was no memory impariment. On other testing with the Thematic Apperception Test, the Rorschach Test, and drawings she showed a good deal of anxiety and inhibition. She showed a lack of childhood spontaneity with related ambivalence in acting out her emotional impulses. It was felt that her ability might well be higher than was demonstrated by the formal testing.

At St. Christopher's Hospital for Children she was found to have a serum phenylalanine level of 20 mg. per 100 ml. Later testing revealed levels between 17 and 25 mg. She has not been given a low phenylalanine diet, and has continued to show normal intellectual capacity.

Discussion

We believe it is important for several reasons to test for phenylketonuria in patients with borderline or even normal intelligence. Occasionally a child with the primary problem of a behavioral disorder might have this disease and thereby be a candidate for treatment. The simplicity of the test would certainly warrant its use in all such children. Secondly, if the diagnosis is made in a person of normal intelligence, subsequent siblings or offspring could be diligently sought out in order to begin treatment at an optimum age. The I.Q. in our younger patient might have been higher had his sister's case been detected before his birth. Finally, the results of treatment can be much better evaluated when we can be sure of the spontaneous rate of normal or borderline I.Q.'s in patients with phenylketonuria.

Summary

1. Two siblings with untreated phenylketonuria who have I.Q.'s of 96 and 73 have been reported. This brings the total number of such patients with borderline or normal intelligence reported to 26.

2. It is important to look for this disease in all mentally retarded patients and in all children with behavioral disorders. Even when intellectual function is normal, it is important to diagnose this biochemical disorder from the standpoint of preventing mental retardation in subsequent siblings or offspring, and of evaluating the efficacy of treatment.

Acknowlegement

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Cytomegalic Inclusion Disease

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Although cytomegalic inclusion disease was first described in 1881, its etiology remained obscure until 1956, when the salivary gland virus was isolated at autopsy from newborn infants with clinical cytomegalic inclusion disease, and from an adult salivary gland. This virus is widespread among animals and humans, but is rarely responsible for pathologic conditions. When it is transmitted to the fetus across the placenta, however, the outcome is usually fatal.

Report of Case

A 20 day old female infant was admitted to Medical Center Hospital in Odessa on May 30, 1960, because of jaundice since birth.

Present illness

The infant was the third child of a healthy 24 year old housewife. The only noteworthy feature of the pregnancy was the fact that the mother had had a bout of "influenza" during the second trimester—an illness characterized by fever and malaise, requiring three days of bed rest. The other children, now aged 3 and 5 years, were normal, healthy infants, with no history of neonatal jaundice. The family history was negative for anemia or jaundice. The mother's blood type was O Rh-positive. She had had no blood transfusions.

This infant was born at another hospital by spontaneous delivery at 40 weeks of gestation; the birth weight was 6 pounds. The baby cried immediately, but was noted to be mildly jaundiced. A few petechiae were scattered over the trunk and extremities, but there was no other evidence of a bleeding tendency. The liver and spleen were not enlarged. The infant showed no evidence of anemia, and the Coombs test was negative.

The jaundice and petechiae, which were described as "mild," improved somewhat during hospitalization. Since the infant was eating well and seemed normal in every other respect, she was sent home from the hospital on the fourth day, weighing 5 pounds, 7 ounces.

The infant did well at home. The mother believed that the stools and urine were normal, and although mild icterus persisted, the baby remained afebrile and active until four days prior to admission. At this time the jaundice increased and more petechiae appeared. The infant became listless and apathetic, and refused feedings. She had had no convulsions.

Physical examination

On admission the weight was 5 pounds 14½ ounces. The head measured 11½ inches in circumference, the chest 13 inches, and the abdomen 14 inches. Temperature, pulse, and respirations were normal. The infant appeared quite ill, being pale, jaundiced, and lethargic. A few petechiae were scattered over the entire body. The anterior fontanel was patent and small. The conjunctivae were icteric but the eyegrounds appeared normal. The heart and lungs were clear. The abdomen was distended, and the liver and spleen could be palpated approximately 4 cm. below the costal margins. The Moro reflex was poor but equal bilaterally. The infant cried weakly on stimulation.

Laboratory studies

The hemoglobin was 13.2 Gm. and the platelet count 112,000; there were 18,850 white blood cells, with 2 promyelocytes, 1 eosinophil, 7 myelocytes, 7 juveniles, 11 stab cells, 12 segmented cells, and 60 lymphocytes. The hematocrit was 39 per cent; the mean capsular hemoglobin content was 32. There were 19 nucleated red blood cells per 100 leukocytes. Urinalysis was negative for albumin, sugar, and urobilinogen, but was positive for bile; microscopic examination showed 7-8 white blood cells, 0-3 red blood cells, and numerous squamous cells. A number of transitional epithelial cells and a few renal tubular cells were seen in the urinary sediment. These large cells contained inclusion bodies which were suggestive of cytomegalic inclusion disease.

The infant's blood was O Rh- positive, like the mother's. Indirect and direct Coombs tests were negative. The stool contained both bile and urobilinogen. A serologic test for syphilis was negative. The total serum bilirubin was 16.9 mg. per 100 ml.—10.6 mg. direct, and 6.3 mg. indirect. Transaminase SGO was 234 Frankel units. Cephalin flocculation was 4 plus in 48 hours. Zinc turbidity was 11 mg. per 100 ml.

A throat culture was negative for pathogens. A spinal fluid examination done on admission showed xanthochromic fluid containing 1 lymphocyte; the total protein content was 180 mg. per 100 ml., sugar 75 mg. per 100 ml.; culture was negative. Chest films showed a flocculant infiltration in the central portions of both lungs fields, thought to represent bronchopneumonia. Enlargement of the liver and spleen was also noted. Roentgenograms of the long bones were normal. A skull film showed calcification scattered throughout the cortex, distributed rather heavily in the basal ganglia; the cranium appeared small, but the sutures were patent.

Course of illness

The infant's condition did not change during her three days in the hospital. Her temperature remained normal and she took her feedings well, although she continued to be listless. The jaundice and petechiae persisted. She was given tetracycline drops, 50 mg. per kilogram of body weight, and was discharged on this medication. At home there seemed to be little change in her condition until she quietly expired three days following discharge.

Postmortem findings

The body was that of a female infant weighing 2,820 Gm. and measuring 48 cm. in length. The head was small, measuring 11.5 inches in circumference. The skin showed generalized icterus, and numerous petechiae were present over all the skin surfaces. The eyes were not remarkable save for the icterus of the sclerae.

The lungs together weighed 70 Gm. and showed a remarkable grayish-red mottling throughout. Microscopic examination revealed an extensive diffuse, interstitial type of pneumonia with heavy infiltration of lymphocytes in the supporting tissues and about many of the bronchi. In multiple sections very large epithelial cells containing huge nuclei were noted. The nuclei contained rounded inclusion bodies, which were stained dark blue.

The liver was markedly enlarged, weighing 258 Gm. The intra- and extrahepatic ducts appeared normal. On microscopic examination a few bile ducts showed large, swollen epithelial cells, in the nuclei of which inclusion bodies were found. There was prominent extramedullary hematopoiesis. The thyroid appeared grossly normal. In a few thyroid acini, however, microscopic examination revealed large, swollen epithelial cells containing large inclusion bodies, a number of which showed the rather typical owl's eye appearance.

The pancreas appeared grossly normal, but also showed, within the pancreatic acini and pancreatic duct, cells containing very typical inclusion bodies.

The kidneys together weighed 34 Gm. and showed the usual configuration. On section very extensive infiltration of lymphocytes was observed in the interstitial tissues, particularly in the cortical zones. A number of the tubules showed large, swollen epithelial cells containing typical owl's eye nuclei in the lining. The cells were more frequent in the convoluted tubules and in the segments of Henle's loop.

The brain weighed 106 Gm. and was less than half the normal size. The cerebral gyri were all hypoplastic, and gross bands of calcification were seen over the surface of the cerebral hemispheres. These were most prominent over the parietal lobes, and extended over the posterior portion of the frontal lobes. The inferior surfaces of the brain were practically free of calcification. On section the calcified areas measured 1-2 mm. in width. Similar calcifications were seen bordering the ventricular system, particularly involving the walls of the lateral hemispheres in the frontal and parietal areas. The intervening portions of the cerebral substance showed no gross calcification. The ventricular system was markedly dilated, with marked enlargement of the lateral ventricles, the foramina of Monroe, the aqueduct, and the third and fourth ventricles. Multiple sections of the brain showed thickening of the pia-arachnoid and rather heavy infiltration of lymphocytes in the meninges. The cortex of the brain was thin, and there were deposits of calcium salts about the areas of lymphocytic infiltration. Heavy calcium deposits were noted in the subependymal areas of the brain.

Discussion

Forms of the disease

When the salivary gland virus is present in adults, it seldom produces recognizable symptoms and usually remains localized in the salivary glands. Occasionally, however, in association with an unrelated debilitating disease of long duration, changes characteristic of those caused by this virus can be found in the lungs, kidneys, intestinal tract, or other organs. Apparently the same course of events occurs in children, since salivary inclusions are found in 10 to 30 per cent of pediatric patients coming to autopsy.

At this time, infection with the salivary gland virus is known to take four forms:

1. Infection of adults, with obscure symptoms and pathologic changes limited to the salivary gland.
2. Infection of children and adults, with spread to the lungs or intestine in association with some unrelated, debilitating disease.
3. Infection of infants and small children involving the lungs and intestinal tract, with death due either to the primary viral infection or to secondary bacterial invaders.
4. Transplacental infection of the fetus, producing a highly fatal, widely disseminated disease.

In a collection of 132 cases of cytomegalic inclusion disease, 32 per cent occurred in newborn infants, 10 per cent as primary pulmonary disease in children, and the remainder in association with other disease processes such as central nervous system disorders, syphilis, and pertussis.

Symptoms and signs

The following signs and symptoms are characteristic of this condition when it occurs in the neonatal period: (1) a small and immature infant, even though the estimated gestation is 40 weeks or more; (2) jaundice; (3) hemorrhagic phenomena, characterized by petechiae and hemorrhage in any viscera or organ systems; (4) hepatosplenomegaly; (5) microcephaly and convulsions; (6) clinical and roentgen evidence of pulmonary disease; (7) anemia, depending upon the extent of the hemolytic and hemorrhagic manifestations; (8) thrombocytopenia; and (9) reticulocytosis.

The virus seems to have a predilection for epithelial cells, though it may be found in endothelium also. Characteristic inclusion bodies are most frequently demonstrated in cells lining the ducts of various organs, as in the present case. An infiltration of lymphocytes, macrophages, and reactive fibrosis often accompanies the inclusion-containing cells, producing cirrhosis in the liver and interstitial pneumonia in the lungs, and sometimes may obstruct the renal tubules. The damaged renal cells slough into the urinary tract and can be demonstrated in the urinary sediment, thus making possible a clinical diagnosis. When the brain is involved, there is gross softening with focal necrosis and mononuclear infiltration: at autopsy calcium deposits are found grossly and microscopically.

When the disease is acquired *in utero,* cytomegalic inclusions are most prominent in the kidneys. When the disease is acquired in infancy and proves fatal, inclusions are found predominantly in the lungs. The carrying effect of the salivary gland virus at different age periods is directly related to the varying susceptibility of the tissues at different ages. It is only in the early, congenital form of the disease that symptoms of blood destruction, with compensatory erythropoiesis, are produced.

Differential diagnosis

Among the conditions occurring in newborn infants, and producing manifestations similar to these of cytomegalic inclusion disease are:

1. Erythroblastosis due to Rh and ABO incompatability, which may be differentiated by blood typing and the Coombs test.
2. Congenital syphilis. This usually can be diagnosed serologically.
3. Acute sepsis. Differentiation would depend largely upon the demonstration of pathogenic organisms by culture.
4. Congenital atresia or absence of the bile ducts. In this situation urinary urobilinogen may be absent and the stools acholic. The direct bilirubin levels increase rapidly. In the majority of these infants the diagnosis is made by exploratory laparotomy.
5. Congenital toxoplasmosis. This may be clinically indistinguishable from cytomegalic inclusion disease since it cause similar intracranial calcifications, chorioretinitis, micro- or hydrocephalus, hemorrhagic phenomena, and hepatosplenomegaly. The dye test is positive in this disease.
6. Congenital hemolytic anemia. There is usually a strong family history for this disease, associated with spherocytosis and abnormal red cell fragility.
7. Congenital leukemia. Though rare, this may be diagnosed by peripheral blood smear and bone marrow aspiration.
8. Hereditary, non-spherocytic, hemolytic anemia. In this disease, the Coombs test is

negative and the osmotic and mechanical fragility tests are normal. There usually is a family history of the disease, however. Unlike congenital hemolytic anemia, this condition does not respond to splenectomy.

9. Generalized herpes simplex infections in the newborn, with resulting hepatitis. Usually, the mother will have some manifestation of infection with herpes simplex virus in the immediate antepartum or postpartum period. Transplacental transmission of this virus has been demonstrated. The infant may show characteristic vascular lesions of the mucous membranes or skin. At postmortem examination inclusions typical of herpes simplex are found, particularly in the liver.

Treatment

No effective therapy for this disease has been found. Steroids and gamma globulin have had little effect on its course. Exchange transfusions produce a temporary decrease in the serum bilirubin, but have no effect on the general course of the disease. Cytomegalic inclusion disease in the newborn is believed to be almost always fatal. With improved diagnostic procedures, there have been recent reports of cases in which the infants survived, but sustained varying degrees of neurologic damage.

Since the salivary gland virus was isolated in 1956, the prevention of this disease has represented a great challenge to the virologist. It is hoped that new tests will be developed to detect the carrier state, particularly in pregnant women, and that eventually, by tissue culture techniques, an effective vaccine will be produced.

Familial Dysautonomia

Report of a Case With Some Unusual Features

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Familial dysautonomia was first reported as a distinct clinical entity in 1949, by Riley and his co-workers[1]. Several other case reports have appeared in the literature since then [2-6], but none from West Virginia.

The case I am reporting presented two features which are apparently unique. The first was a rectal temperature of 110 F., with the patient surviving. The second unusual feature was the subsequent development and persistence of a curious jaw spasm which makes it impossible for the patient to close his mouth for periods of time varying from 30 minutes to two hours.

Case Report

The patient is a 14 year old, Jewish male who has been under my care for the past six and a half years. He has, at one time or another, exhibited most of the "classical" findings of familial dysautonomia: defective lacrimation, vascular disturbances, skin eruptions, excessive perspiration and salivation, and dysfunction of the respiratory and digestive systems. Both his parents are of Russian Jewish extraction, although there is no consanguinity. His only brother, aged 20, is perfectly normal.

Past history

The mother's pregnancy, labor, and delievery were normal, and the patient weighed 6 pounds and 9 ounces at birth. Soon after birth he became a problem eater; he appeared to swallow with difficulty, and his weight gain was extremely slow; at 2 years of age he weighed only 20 pounds. During infancy he had many breath-holding spells, which were a source of great concern to his parents. This boy's motor development was extremely retarded. He sat up at 18 months, walked at 4 years, and talked at 3 years. His parents were most alarmed, however, by the fact that he was never able to shed tears, and for this reason he was referred to the John Hopkins Hospital in June of 1949, when he was 3 years old. Here

---

From the Beckley Hospital, Beckley, West Virginia.

no definite diagnosis was made. When the patient was 5 years of age, a corneal ulcer developed on the right eye. He was referred to the Babies Hospital in New York, where Dr. Conrad Riley made the diagnosis of autonomic dysfunction, or familial dysautonomia.

The patient has had at least eight known cases of bronchopneumonia (confirmed by roentgenogram). Seven of these episodes occurred prior to 1956; the last was in 1958. In addition, he has had 26 admissions to the Beckley Hospital because of persistent vomiting, with resultant dehydration and metabolic acidosis. He usually responds well to parenteral fluids, and chlorpromazine and Sodium Seconal, and is usually discharged within two to five days. These episodes have occurred in spite of the fact that for the last two years he has been maintained on daily prophylatic doses of chlorpromazine and meprobamate. His emotional lability, which has always been a problem, is becoming worse with each hospital admission.

Symptoms and signs

When I first saw this patient in 1954, the diagnosis of familial dysautonomia had already been made. Among the vascular disturbances manifested by this patient are hypertension with excitement (a diastolic pressure as high as 110 has been recorded, with a systolic pressure of 160), and postural hypotension (80 systolic, 50 diastolic). When he is unduly tense or excited, a blotchy eruption appears on his face and body, and occasionally on his extremities; this disappears within minutes. His hands and feet are always cold. He perspires excessively, and still drools. He is usually constipated, though he has had diarrhea on several occasions.

The patient's coordination is poor, and his gait is slightly ataxic. He has a very pronounced kyphoscoliosis. His deep tendon reflexes are sometimes absent and sometimes hyperactive. So far he has had no classical convulsions, but repeated electroencephalograms have been diffusely abnormal; the usual findings have been a diffuse dysrhythmia of the fast type, plus evidence of subcortical and more diffuse cortical or parasagittal involvement. Recent electroencephalograms have showed, in addition, more frequent three-per-second spikes, and waves occurring in bursts and diffusely. The alpha activity is not blocked by painful stimuli.

Until recently the patient seemed relatively insensitive to pain. He has a deformity of the left elbow which is the result of an unrecognized fracture sustained when he was 4 years of age. Lately, however, he has shown a tendency to hyper-reaction when painful stimuli are applied; he complained of severe pain when given a polio injection with a 25-gauge needle.

Laboratory data

Except during the episode of extreme hyperpyrexia, all blood chemistry determinations, blood counts, urinalyses, serologic and sweat tests have been within normal limits. Chest films show kyphoscoliosis, most marked in the mid-thoracic spine, and pulmonary changes consistent with bronchiectasis in the right middle lobe and possibly in the medial segment of the right lower lobe. Since no clinical abnormality is present now, no further roentgenograms of the chest have been made. Films of the left elbow show severe osteoporosis of all bones in that area, with atrophy of disuse.

Episode of hyperpyrexia

Between 1954 and 1956 the patient was admitted ten times to the Beckley Hospital: six times because of bronchopneumonia, and four times because of high fever and vomiting. The temperature upon admission was usually 105 and 107 F.; on one occasion it was 108. On December 29, 1956, at 11:30 P.M. he was admitted in a semicomatose condition. Characteristically, he had been well until eight hours before admission, when he became feverish and soon afterwards began to vomit. On admission the rectal temperature, taken by a registered nurse, was 110 F.; a few moments later I verified this reading with a different thermometer. The systolic blood pressure was 50.

We immediately put cold wet towels over his body, and then placed him in a bathtub of cold water. An infusion of 5 per cent glucose and water, with Levophed, was started in his left ankle. He was given 200 mg. of Solu-Cortef and 1,000,000 units of aqueous penicillin intravenously, followed by intramuscular injections of Solu-Cortef, 100 mg., and penicillin, 1,000,00 units, every four hours. Chloramphenicol was also given intramuscularly every four hours in doses of 250 mg.

By 7 A.M. on the following day, the patient's temperature was down to 100 F. rectally; his blood pressure was 120 systolic, 70 diastolic, and he was able to take fluids by mouth. He was rational and cooperative. Examination of spinal fluid showed normal findings: a cell count of 0; 45 mg. of sugar, 35 mg. of protein, and 720 mg. of chloride per 100 cc.; and no bacteria on the smear. The hemoglobin was 10.5 Gm., and there were 10,650 white cells, with 3 eosinophils, 46 lymphocytes, 1 monocyte, and 50 segmented cells. Urinalysis showed a 2 plus reaction for albumin and an occasional white blood cell; subsequent urinalyses, however, were normal. A roentgenogram of the chest was essentially unchanged from films taken three months previously, following complete recovery from an episode of bronchopneumonia.

All medications were discontinued within 12 hours after admission. The patient remained afebrile, and was discharged on the fifth hospital day, with no obvious untoward effects. It is interesting to note that the highest temperature observed in this patient subsequently was 105 F., with his last attack of bronchopneumonia in 1958. Recently, his temperature has not been above 102 F.

Jaw spasm

Thirteen months after this episode of hyperpyrexia, the patient was unable to close his mouth for a period of almost two hours. Since then, episodes of jaw spasm have occurred on an average of two to three times per month. During these episodes the mouth is kept wide open, and although the patient is perfectly rational, he is unable to close it for periods ranging from one-half hour to two hours. Examination and roentgenographic studies of his jaws by orthopedists at our hospital and at the Babies Hospital in New York have disclosed no local abnormality. The current impression is that these episodes are probably epileptic equivalents, and perhaps the result of brain damage initiated by the hyperpyrexia. So far we have not succeeded in preventing these episodes by the use of phenobarbital, Dilantin, Milontin, Mysoline, or Tridione, but we have discovered that they can be terminated by the prompt intramuscular injection or 1½ grains of Sodium Seconal. In spite of all reassurances, these episodes terrify the patient.

Progress note

This boy is still markedly underweight (52 pounds) and short for his age, measuring 50 inches. Physically, he is a pathetic specimen. Mentally and socially, however, he functions surprisingly well between hospital admissions. In spite of the many illnesses and the psychic trauma he has suffer ed, he is not mentally retarded at the present time. His last psychometric evaluation (three years ago at the Babies Hospital in New York) placed him within the "dull normal range." He is now receiving instruction at home, and will soon be promoted to the sixth grade. He is a happy boy and, his parents inform me, quite shrewd. He has a paper route (he insisted on this), and was recently chosen as "the newspaper boy of his county." The brightest spot in his outlook is that he is generally cheerful and optimistic. In the light of his history, this disposition might be considered the most unusual aspect of his case.

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OCTOBER, 1961

DR. WILBURT C. DAVISON

Months ago the Editorial Board of the NORTH CAROLINA MEDICAL JOURNAL voted to devote the October issue to commemorating the long and invaluable service rendered by Dr. Wilburt C. Davison since he came to North Carolina as Dean of the state's first four-year medical school. Tributes to him by Drs. Nicholson, Arena, Andrews, and others speak for themselves. Since, however, the Editor and Assistant Editor are the only ones participating in the preparation of this issue who were not former students, house officers, or faculty colleagues of Dr. Davison, it is our privilege to add our own personal message.

One of the most noteworthy features of assembling material for this issue was the admiration, appreciation, and affection displayed by the contributors. Although this number contains perhaps the largest aggregate of original articles yet published in this *Journal's* history, it could have been twice as large as it is. It was necessary to set a limit on the number of papers accepted—hence many had to be declined. To those who were disappointed because their papers could not be included in this tribute to their beloved friend and teacher, we offer our regrets, for we know that they really wanted to join in the chorus of appreciation. We are sure that Dr. Davison appreciates and reciprocates the affection lavished upon him by those who were not included in this *Festschrift* as much as he does in the case of those whose names do appear.

Dr. Davison, the Editorial Board, on behalf of the doctors of North Carolina, salutes you and honors you for what you have meant to medical education and to medical practice in North Carolina, the nation, and the whole world. You have left an indelible impression on all who have known you. May your wholesome influence live on and on in the years to come.

* * *

A WORD OF EXPLANATION

It will be noted that a number of departments which appear regularly in this JOURNAL have been omitted from the present issue, or sharply curtailed. This was done in order to devote as much space as possible to the tributes and scientific contributions, and to hold extraneous material to a minimum.

Because of the unusual length of the bibliographies accompanying some of the papers, references were omitted in a number of articles—also to save space. In such cases, the references were set in type and will be included in the author's reprints.

* * *

ASTRONAUT AND ANTHROPOID

For the first time a human being has put a girdle round about the earth. The 25 hours 18 minutes required for 17 circles around the globe presents an average of not q 90 minutes for each complete circle. E though this is a little more than twice the time Shakespeare's Puck said he would require, Major Titov deserves world acclaim for his feat. An editorial in the *News and Observer* for August 8 says so well what needs to be said that it is quoted in full:

> The world's admiration is certainly due the Russian scientists who have put men in orbit around the earth and similar scientists in America who are not far behind them in this dramatic enterprise. Involved in this matter is not merely the competition of nations but also the advance of mankind in knowledge and power. The very brilliance of the achievement, however, tragically emphasizes the lag in human intelligence beside the thrust forward in human skill.
>
> We are beginning to probe space before we have learned to make order and decency, peace and plenty on the planet which has become a launching pad into the universe. Yet so far as "science" is concerned and the energy and treasure with which to equip it, we have every power to save, house, feed, educate all the people of the earth, not in millenium, but now. In shooting at the moon, the greatest nations are turning their backs on man.
>
> In an age of astronauts, we are still predatory anthropoids. We have come down from the trees and now are headed toward the stars. Sometimes in what are described as diplomatic conferences, we still seem bickering, gibbering apes. Increasingly we have learned how to deal with every aspect of nature but ourselves.
>
> Man and not merely the Russians or the Americans have a right to pride in the scientific achievements of this generation which can control a manned capsule in the cosmos. The very brilliance of the achievement, however, tragically emphasizes man's failure to learn—apparently even begin to learn—to control man for his own safety and well-being.

* * *

AN ALL-EMERITUS FACULTY

The following editorial from the *New York State Journal of Medicine* for July 15 is so pertinent that it is quoted in full.

In 1948 the University of California's Hastings College of Law, perennially faced with the problem of building a high level faculty, adopted the policy of appointing to its full-time staff only men sixty-five years of age or over. The Hastings faculty is made up of distinguished scholars and teachers retired by other law schools. It has grown to become the largest law school west of St. Louis, and its graduate body has one of the highest records of achievement in the country. The outstanding success of Hastings, attributable largely to its sixty-five and over faculty recruitment, demonstrates the ability of older professional persons to continue to make an important contribution as teachers.

This experience adds evidence to the kind of opportunities that exist for more effective utilization of older professional and scientific personnel.

Our municipal and state hospitals are in the dire straits that Hastings was in thirteen years ago. Could not a similar principle of enlightenment be applied? The recruitment of medical talent where retirement has been mandated could be a profitable venture for all concerned. For the institution there would be a seasoned, knowledgeable nucleus whose leadership would draw the apprentices, who in turn would fill the gaps in service. For the individual there would be that prolonging of usefulness so important to the deceleration of life.

We are as wasteful and heedless of our human resources as we are of our material resources. In the face of overriding need we should use our medical substance to the full.

* * *

PEARLS AS TRANQUILIZERS

A double-column advertisement in the *New York Times* for Sunday, June 18, adds one more tranquilizer to the growing list. This new tranquilizer is fairly expensive, prices ranging from $39.75 to $175.00. According to the advertisement, however, it may well be worth the money. The advertisement states that "No sedative can make you feel more relaxed and poised, No facial can make you feel more chic and glamorous, No psychiatrist can make you feel more secure about your own loveliness than a fabulous new OPERA LENGTH NECKLACE OF EXQUISITE CULTURED PEARLS."

The name and address of the advertiser will be furnished to any interested psychiatrist.

Bulletin Board

COMING MEETINGS

Fifth District Medical Society Meeting—Carolina Hotel, Pinehurst, October 11-12.

Association of American Physicians and Surgeons, Annual Meeting—Grove Park Inn, Asheville, October 12-14.

American Cancer Society, North Carolina Division, Annual Meeting—Sir Walter Hotel, Raleigh, October 14-15.

The Charlotte Postgraduate Seminar—Presbyterian Hospital, Charlotte, October 18-19.

North Carolina Neuropsychiatric Association Meeting—Raleigh, October 27.

University of North Carolina School of Medicine, Fifth Annual Symposium on Infectious Diseases—University of North Carolina School of Medicine, Chapel Hill, November 9-10.

North Carolina Pediatric Society Annual Meeting—Sedgefield Inn, Greensboro, November 10-11.

North Carolina Academy of General Practice, Annual Meeting—Carolina Hotel, Pinehurst, November 26-29.

North Carolina Mental Health Association, Annual Meeting—Jack Tar Hotel, Durham, February 16-17, 1962.

Nineteentth Annual Watts Hospital Medical and Surgical Symposium—Watts Hospital and Jack Tar Hotel, Durham, February 23-24, 1962.

Southeastern Allergy Association, Annual Meeting—Thomas Jefferson Inn, Charlottesville, Virginia.

Southern Medical Association, Annual Meeting—Adolphus Hotel, Dallas, Texas, November 6-9.

Association of American Medical Colleges, Seventy-second Annual Meeting—Queen Elizabeth Hotel, Montreal, Canada.

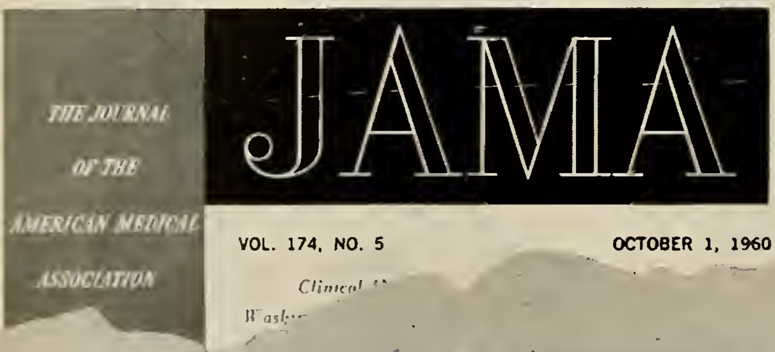
American Medical Association Clinical Meeting—Denver, Colorado, November 27-30.

Southern Surgical Association, Annual Meeting—The Homestead Hotel, Hot Springs, Virginia, December 5-7.

NEW MEMBERS OF THE STATE SCOIETY

Dr. Loyd Clarence Brannon, 1321 Canterbury Road, Raleigh; Dr. Lewis James Gaskin, 1330 St. Mary's Street, Raleigh; Dr. Joseph Lee Sedwitz, Zebulon; Dr. Carlton Dbu Everhart, 48236 Bialas Street, Selfridge, AFB, Michigan; Dr. Richard Bruce Patterson, 1550 Drumcliffe Road, Winston-Salem; Dr. Winfield Hardy, Mt. Sanitarium Hospital, Fletcher; Dr. Edwyn Taylor Bowen, Jr., 2240 Cloverdale Ave., Winston-Salem; Dr. William Robert Purcell, 410 King Street, Laurinburg.

Dr. Campbell W. McMillan, 410 King Street, Laurinburg; Dr. William Ernest Bakewell, Jr.,



newest J.A.M.A. paper[1] *reports*

DBI

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N. C. Memorial Hospital, Chapel Hill; Dr. Donald Carlisle Abele, N. C. Memorial Hospital, Chapel Hill; Dr. Gustin Thomas Kiffney, Jr., N. C. Memorial Hospital, Chapel Hill; Dr. Claude Raymond Nichols, Duke University Medical Center, Durham; Dr. Allen Dale Smith, 1200 Broad Street, Durham; Dr. John Laszlo, Duke Medical Center, Durham.

1961 MARCH OF DIMES SCHOLARSHIP WINNERS

Thirteen North Carolina students have been awarded 1961 National Foundation health scholarships, according to Basil O'Connor, president of the March of Dimes organization.

Each year more than 500 scholarships are offered nationally to qualified young Americans seeking careers in medicine, nursing, physical therapy, occupational therapy, or medical social work. Each scholarship is for $500 a year for four years of college or university education.

North Carolina scholarship recipients announced today are:

Nursing: Ethel Marie Griffith, Winston-Salem; Dorothy Rose Isom, Raleigh; Constance Lee Newnam, High Point.

Occupational Therapy: Betty Anne Long, Wallace; Nancy Carol Moss, Martha Eloise Styles, Asheville.

Physical Therapy: Juanita Christian Caldwell, Greensboro; Sandra Lee Lepscier, Murphy; Susan Mary Matuszak, Winston-Salem.

Medicine: Robert Lee Grubb, Jr., Charlotte; Margaret Louise Craig Johnson, Fuquay Spring.

Medical Social Work: Rosebud Margaret Richardson, Wilmington.

Winners are selected in each state by a committee composed of state health leaders representing the five professions included in the program.

EDGECOMBE-NASH MEDICAL SOCIETY

The Edgecombe-Nash County Medical Society held its regular monthly meeting on September 13.

The speaker was Dr. J. R. Hester of Wendell.

AMERICAN MEDICAL ASSOCIATION

American physicians are preparing this fall to move into the forefront of a concerted campaign against mental illness and for better mental health for all.

As an opening step in the campaign, a preliminary planning conference, was held September 29-October 1 in Chicago to begin work on a specific program.

The conference sought to identify areas and methods in which physicians and the A.M.A. can provide additional support to catalyze the de-

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1. Barclay, P. L.: J.A.M.A. 174:474, Oct. 1, 1960.

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velopment of programs for community and hospital services, research, and in professional and lay education.

The program developed will later be presented to mental health representatives of state medical associations for their consideration and discussion. This group is tentatively scheduled to meet in January or early February.

One of the first major activities of the new mental health campaign will be to hold the First American Medical Association Congress on Mental Health, to be held in cooperation with the American Psychiatric Association. The congress is tentatively scheduled for June, 1962, in Chicago. Through the congress, the A.M.A. will provide a forum to which both individuals and representatives of agencies and organizations interested in any of the aspects of mental health will be invited.

Leo H. Bartemeier, M.D., of Baltimore, is chairman of the A.M.A.'s Council on Mental Health. M. Ralph Kaufman, M.D., of New York City, is chairman of the planning committee.

NEWS NOTES FROM THE DUKE UNIVERSITY MEDICAL CENTER

Symposium Honors Dr. Davison

Three internationally known figures in education, philosophy, and health were keynote speakers for Duke University's Symposium on the Commonwealth of Children held October 4-8.

They were Dr. Grayson L. Kirk, president of Columbia University; Dr. George Boas, professor emeritus of philosophy at the Johns Hopkins University; and Dr. Brock Chisholm, former director-general of the World Health Organization.

The symposium was conceived as a tribute to Dr. Wilburt C. Davison, dean of the Duke Medical School from its beginning in 1930 until he relinquished the post a year ago. Dr. Davison retired from the School's pediatrics faculty this summer.

Dr. Jerome S. Harris, chairman of the Department of Pediatrics, was chairman of the Symposium.

* * *

New Child Care Facilities Opened

New facilities that mark a major forward step in providing the best possible care for infant and child patients at the Duke University Medical Center have just opened.

An addition to Howland children's ward has increased the number of pediatric beds from 33 to 50. The original ward has been completely reconstructed and tied in with the addition. Both the new and older areas are air conditioned.

Total cost of the project was $440,000. The money came from Duke University funds, the Doris Duke Foundation, the North Carolina Medical Care Commission, and the Hospital and Orphans Section of the Duke Endowment.

Dr. Jerome S. Harris, chairman of the Duke pediatrics department, said the project was necessitated in part by the child population increase since World War II. He noted also that Duke Hospital serves North Carolina and many other states as a referral center.

"Many of our pediatric patients are referred to Duke because they present difficult diagnostic problems or require very specialized treatment procedures," Dr. Harris said. "Special facilities and a high ratio of personnel to patients must be maintained in order to care properly for these children."

Dr. Harris noted that in addition to patient care facilities, the new ward offers better provisions for training graduate nurses to become specialists in pediatric nursing, and for coordinating the activities of physicians, nurses, physical therapists and others in rehabilitation work with child patients.

* * *

Duke Cited for Medical Research

Medical research at Duke University has been cited by the Southern Regional Education Board in its publication, "University Research—Medical Milestones," which has just been released.

Mentioned specifically was research dealing with hypothermia, or artificially lowered body temperature.

Duke scientists "have done pioneer work in the use of extremely low body temperatures as a means of increasing the patient's safety and simplifying certain surgical procedures during open heart operations," the booklet states.

"With a special blood-heat exchanger, surgeons at the Medical Center have lowered the body temperature of surgical patients to approximately 50 degrees Fahrenheit below normal."

The Southern Regional Education Board reports that all over its 16-state region, scientists in university laboratories are studying man and his ills to determine what direction progress will take in the future.

The SREB publication is one of a series of informational brochures on university research in the South produced under a $5,000 grant from the Sperry and Hutchinson Company.

The Board notes that Southern universities and medical schools are steadily building up research facilities "to handle the future growth of current research projects and the onslaught of new demands in medical research."

Since World War II, new or expanded medical schools have been built in North Carolina, Florida, Kentucky, Mississippi and West Virginia

EASTERN PSYCHIATRIC RESEARCH ASSOCIATION

On Friday and Saturday, October 27 and 28, 1961, a unique interdisciplinary symposium on "Expanding Goals of Genetics in Psychiatry (1936-1961)" will be held in the eighth floor auditorium of the New York State Psychiatric Institute, 722 West 168th Street, New York City, in conjunction with the Sixth Annual Meeting of the Eastern Psychiatric Research Association. With many outstanding participants from the biologic and behavioral sciences, and with Dr. Franz J. Kallmann as the program chairman, this symposium is arousing wide interest among professional groups in the mental health field.

The entire symposium will be held under the auspices of the New York State Department of Mental Hygiene and the Department of Psychiatry, College of Physicians and Surgeons, Columbia University, New York. The program has been arranged in such a manner that the functions and future objectives of a gentics department in the psychiatric area will be assessed together with a review of past and present activities in psychiatric and behavioral genetics and with an authoritative appraisal of recent advances in basic genetics. Cosponsors for this event include: The Academy of Psychosomatic Medicine, The American Eugenics Society, The American Psychopathological Association, The American Society of Human Genetics, and The Scottish Rite Committee on Research in Schizophrenia.

Book Reviews

The House of Healing. By Mary Risley. 288 pages. Price, $4.50. Garden City, New York; Doubleday & Company, Inc., 1961.

This book is primarily a history of hospitals, necessarily including considerable material on medical and general history if the two can be separated. Classification hospitals and the effect of Listerism. Illustrations are few, but of excellent quality.

The fact that there is little in journals or tests on the history of hospitals probably reflects the difficulties of organizing the material in the face of changing concepts of what a refuge for the sick should do. With this in mind, Mrs. Risley may be said to have done creditably. As she is well aware, more questions are raised than are answered, since direct evidence about much of the matter treated is wanting.

The book is suitable for both general and medical libraries, although the absences of a bibliography severely limits its utility in the latter. The treatment is best suited to the lay reader, or the medical reader in search of a short discussion of high lights. Within these limits it is recommended.

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Essentials of Neurosurgery for Students and Practitioners. By Sean Mullan, M.D. 273 pages. Price, $6.75. New York: Springer Publishing Company, Inc., 1961.

Any teacher of neurosurgery is required, at one time or another, to recommend a suitable book to the medical student. Dr. Mullan's book serves this purpose very well. It is written concisely and in such a way that the hardest subjects are made easy. The arrangement of the subject-chapters is unique and unorthodox, but helpful. There is a rather long clinical description of clinical syndromes associated with brain tumors which is valuable in understanding the symptomatology of localized brain disease.

The diagrams are simple and useful; however, this reviewer believes that some of them have been so oversimplified that they may be misleading. For instance, the direction of the x-ray beam in the anteroposterior (Towne's) view should be at an angle rather than perpendicular to the brain. The student might recognize the temporal horns on the x-ray more readily if they were shaped accordingly in the diagram rather than indicated by dots.

In the interest of conciseness, the author had to be rather dogmatic on several controversial subjects, such as the value of lumbar puncture in spinal cord injury.

These minor criticisms should detract in no way from the excellence of the book. It is recommended for the medical student as well as for the resident who is beginning his work in neurosurgery.

In Memoriam

The members of Caldwell County Medical Society were saddened by the death of one of its members, Frederick Mast Dula, on June 23, 1961. His death will be felt deeply by patients, friends, and professional colleagues.

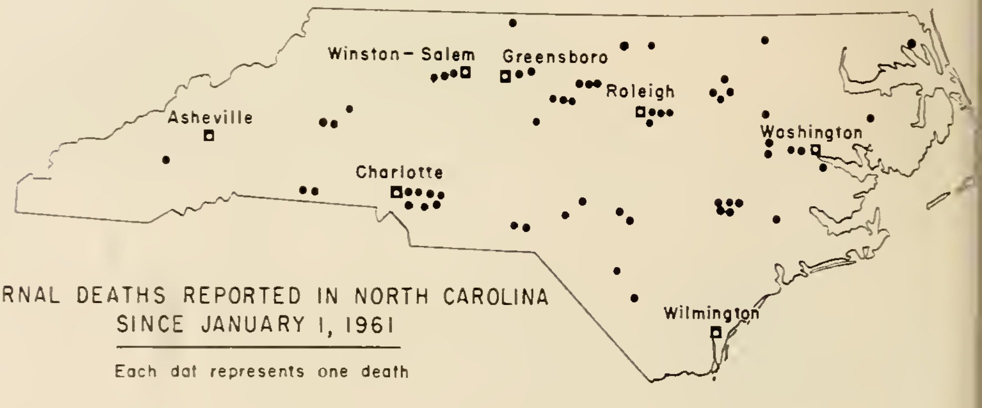
The son of the late Dr. Alfred W. and Mrs. Mary Adeline Mast Dula, he was born in Lenoir, North Carolina, where he attended the graded schools. He went on to obtain a Bachelor of Arts degree and a Master of Arts degree in psychology and later, the degree of Doctor of Optometry. He practiced optometry for five years. Thereafter he entered the Medical School of the University of North Carolina and obtained his M.D. degree from Vanderbilt University in 1932. He interned in Norfolk General Hospital. He returned to Lenoir where, in 1935, he organized and built the Dula Hospital, in which he practiced until his death.

Dr. Dula had many talents and interests. He served in the U. S. Naval Reserve and as a captain saw much active duty during World War II. He was active in civic affairs, and served on the Lenoir City Council for several years. He was closely associated with the work for the Blind and with the Cancer Program in Caldwell County.

He was an active member of several professional groups—including the Caldwell County Medical Society, North Carolina State Medical Society, and the American Medical Association.

Since this Community and County Medical Society has lost a valued member, and each of us a professional friend,

Be it therefore resolved, that we, the members of Caldwell County Medical Society, express our deep sorrow and extend sympathy to the family of Dr. Frederick Mast Dula, and that a copy of these resolutions be placed in the permanent files of the Society, a copy sent to his family, and a copy sent to the Journal of the North Carolina State Medical Society.



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Medical Journal

MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

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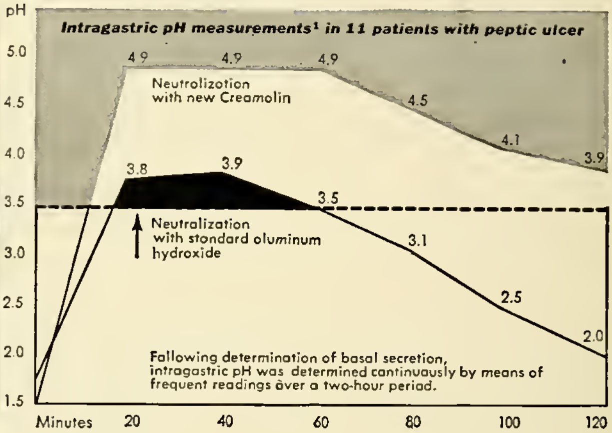

Intragastric pH measurements[1] in 11 patients with peptic ulcer
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Neutralization with new Creamalin
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VOLUME 22 NOVEMBER, 1961 NUMBER 11

Symposium on Anesthesia in the Geriatric Patient

The Preanesthetic Evaluation and Preparation of the Geriatric Patient

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WINSTON-SALEM

In assessing the risk of anesthesia and surgery in the geriatric patient, it is important to determine how much, if any, the functional capacity of his organ systems has been impaired by the degenerative changes of advancing age. Before he is subjected to operation, these systems must be restored to their optimal capacity.

There are many drugs at our disposal which may be used to improve functional capacity. Certain drugs, however, may produce impairment instead of improvement. Too often anesthetic difficulties are ascribed to the patient's inability to tolerate the anesthetic agent. In general, most patients tolerate anesthesia well, with suitable preparation and proper management.

It is often assumed that measures have been instituted to provide optimal conditions for anesthesia and surgery. Nevertheless, the anesthesiologist has a major responsibility in assessing the preoperative preparation and in planning the most appropriate form of anesthetic management for each patient. He should decide whether further measures are indicated to improve the patient's condition in the time available. To correct during anesthesia and surgery errors of omission and commission belonging to the preanesthetic preparation is haphazard management.

Interference with vasomotion, vasoconstriction, and vascular reactivity to epinephrine or norepinephrine in the peripheral vascular bed will result in peripheral vascular dilatation and pooling of blood. The subsequent decrease in venous return will cause a reduction in cardiac output. Compensatory readjustment is dependent on both neurogenic and humoral factors. Interference with compensatory adjustment of the vascular bed may result from preanesthetic drug therapy, hypoventilation, acidosis, or electrolyte and hormonal imbalance.

Problems Associated with Preanesthetic Drug Therapy

In recent years the multiplicity of drugs being given for various conditions has become a new and insidious hazard for surgical patients. Anesthesiologists are faced with the problem of maintaining circulatory homeostasis in patients who have been on long-term therapy with cortisone, antihypertensive preparations, phenothiazine derivatives, or other less potent tranquilizers. When the patient is subjected to the stress of anesthesia and surgery, these drugs often adversely affect the circulation.

Cortisone

With the present widespread use of cortisone, inevitably some patients will face the stress of anesthesia and surgery with suppressed adrenocortical function. The suppressive effects of this drug may last for many months. Adrenocortical insufficiency subjects the patient to severe hypotension, respiratory depression, and delayed recovery from anesthesia. There is some good ex-

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Presented before the Section on Anesthesia, Medical Society of the State of North Carolina, Asheville, May 9, 1961.

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perimental evidence that in adrenocortical insufficiency, vascular responsiveness to circulating catechol amines is diminished. It has been postulated that the loss of sodium and the ensuing distortion of the intracellular-extracellular electrolyte gradient of the vascular muscle cells lowers the blood pressure by interfering with the normal vascoconstrictive efficiency of intrinsic norepinephrine and epinephrine[1].

It is therefore recommended that any patient who has had therapeutic doses of cortisone for more than a week within the six-month period preceding operation should be given supplementary doses the day before, during, and in the period immediately after the operation, in order to prevent acute adrenocortical insufficiency. On the night before the operation 100 mg. should be given. On the operative day the dose should be equivalent to the amount produced by maximal stimulation of the adrenal cortex, which is approximately 10 mg. per hour. This can be administered intramuscularly in a 100-mg. dose, followed by 50-mg. injections every six hours. An alternate method is to administer 100 mg. per liter of 5 per cent dextrose in water by intravenous drip every eight hours. Starting on the first postoperative day, the total daily dosage of cortisone may be reduced in increments of 50 mg. per day.

Antihypertensive agents

Severe hypotension associated with bradycardia may occur in patients who have received rauwolfia derivatives, such as reserpine, for the treatment of hypertension[2]. This untoward effect is related to depletion of the intrinsic catechol amines in the myocardium, vascular wall, and adrenal medulla[3]. Under normal conditions, stores of epinephrine and norepinephrine in the myocardium and vascular wall are released in small amounts, and are necessary as humoral agents for the regulation of the cardiac pacemaker and to maintain a normal state of myocardial and peripherovascular contractility. Correction of the hypotension might logically be obtained by supplying the necessary amount of the missing catechol amine, norepinephrine, or by the use of methoxamine and phenylephrine, which act directly on the vascular wall to produce vasoconstriction. The group of sympathomimetic amines, such as ephedrine, which lose their effect following reserpine therapy, normally act by releasing epinephrine or norepinephrine from the store in the artery wall. The rauwolfia derivatives appear to exert a pharmacologic effect for approximately two weeks after therapy has been discontinued. To avoid undue operative risk, the drug should be discontinued two weeks prior to elective surgery.

Guanethidine (Ismelin), a recently introduced antihypertensive agent, acts by inhibiting the release of norepinephrine from the postganglionic sympathetic nerve endings. The drug has a prolonegd effect and should be discontinued four days prior to anesthesia.

The hazards induced by chlorpromazine and other phenothiazine derivatives lie in their ability to depress or abolish reflex circulatory control. Previous therapy with these autonomic suppressants may be associated with hypotension, tachycardia, and delayed recovery in the anesthetized patient. The phenothiazines can initiate an extrapyramidal syndrome. Coma in the tranquilized diabetic patient may be more precipitous and more prolonged.

The principal danger of chlorthiazide is serum potassium depletion and sodium depletion in the peripherovascular wall. The hypokalemia will enhance the toxic effects of digitalis. The depletion of sodium in the vascular wall will reduce vascular reactivity to circulating catechol amines and other vasopressors[4].

Others

Digitalis, an important drug in preparing many cardiac patients for operation, can be exceedingly dangerous and toxic in excessive amounts. Rapid digitalization just before operation often causes digitalis intoxication, especially if there are uncorrected electrolyte imbalances with low levels of blood potassium.

Curare should be used with caution in the patient on quinidine therapy. Quinidine has been shown to have a curariform action on neuromuscular transmission. It also interferes with the anticurare effect of neostigmine.

The elderly patient is much more sensitive to sedative drugs and narcotics. There is no doubt that older patients have died in the operating room as a result of marked depression from preoperative doses of narcotics such as morphine or meperidine.

Iproniazid (Marsilid), a monoamine oxidase inhibitor, was first introduced as an antituberculous drug, but has also been found to be an effective psychic antidepressant and antihypertensive agent. Meperidine should not be administered to patients taking iproniazid. This combination of drugs may cause agitation with hyperactivity, or coma, hyperflexion, and periodic respiration. Prednisolone given intravenously in 25 mg. doses reverses this picture almost immediately[5].

Blood Volume

An adequate blood volume should be established before anesthesia and surgery. It may be generally stated that no patient with a hemoglobin level below 10 mg. per 100 ml. or a hematocrit below 35 per cent should have an elective operation without correction of the anemia. It should be emphasized that the debilitated patient may have a significantly reduced blood volume even though his hemoglobin and hematocrit levels are normal. Hypovolemia is common in the elderly, chronically ill patient, and in the patient with carcinoma or ulceration of the gastrointestinal tract. Anesthesia, with its associated peripheral vasodilatation, may unmask a chronic hypovolemia. When the blood volume is already diminished, the loss of even small amounts is poorly tolerated, and the patient tends to go into shock more readily. Once hemorrhagic shock has developed, the response to blood replacement is not always prompt. It takes less blood to prevent this emergency than it does to treat it. The patient with hypovolemia may not be able to compensate for changes in body position or for the central nervous system depression required for anesthesia. The result may be profound hypotension from what apears to be a minimal cause.

An attempt should be made to correct the deficit in blood volume before the induction of anesthesia in order to avoid the consequences of shock. Evaluation to determine the cause of the anemia or the reduced blood volume, and the institution of appropriate therapy, are preferable to risking a transfusion reaction. Except in an emergency, iron deficiency anemias are usually more appropriately corrected with iron therapy. Few situations are so urgent that time cannot be taken to institute measures to restore an adequate volume of circulating blood. With the transfusion of whole blood, plasma dispersal is essential to raise the hematocrit. Adequate time is necessary for this to be done by the patient's own renal and metabolic mechanisms. Twenty-four hours without transfusions should be allowed for equilibration prior to anesthesia. The elderly patient with heart, liver or renal disease requires even longer. The use of packed red cells will obviate the necessity of plasma dispersal. This is important in emergency situations, in patients whose plasma-dispersal mechanism is impaired, and where the danger of producing hypervolemia exists. Preoperative transfusion of whole blood immediately prior to surgery may subject the elderly patient to hypervolemia, with subsequent cardiac decompensation and pulmonary edema.

Cardiac Function

The patient's specific type of cardiac disease is less important than the effective function of the heart. The elderly patient may show a diminished cardiac reserve even though symptoms are absent. Arteriosclerotic changes of varying degrees are a constant accompaniment of advancing age. These changes affect both the myocardium and valves as well as the coronary circulation.

Angina pectoris and coronary artery disease are a common cause of sudden death in the operating room. The surgical risk is greatly increased after a recent myocardial infarction. If the infarction has occurred within the past three months, the operation should be delayed, if possible. This is the period of time generally accepted as that required for satisfactory healing. Increasingly severe attacks of angina on progressively less exertion is a warning of impending coronary occlusion, and operation should be avoided, if possible.

The senile heart is less able to compensate for circulatory stress by increasing cardiac output through an increase in myocardial contractile force and heart rate. Anesthesia with its associated myocardial depression and peripheral vasodilation may be sufficient to unmask incipient myocardial decompensation.

The elderly heart is more prone to develop abnormal rhythms. Certain arrhythmias appear more significant to the anesthesiologist than do others. The patient with chronic atrial fibrillation whose ventricular rate is well controlled with a digitalis glycoside usually does well during anesthesia. However, extreme caution should be taken with the patient having a history of episodes of acute atrial fibrillation. The onset of atrial fibrillation during operation is a warning of impending cardiac decompensation, and the operative procedure should be terminated as soon as possible.

Premature atrial or ventricular contractions may be the forerunner of incipient cardiac decompensation or more ominous arrhythmias.

Heart block is usually of more serious import. A well established bundle branch block does not appear to greatly enhance the risk of surgery. The patient with complete atrioventricular block, however, presents an extremely grave risk for anesthesia and surgery. These patients are subject to Stokes-Adams attacks and sudden cardiac arrest during operation. Prophylaxis consisting of atropine sulfate, 1 mg. given intramuscuarly one hour preoperatively and 0.5 mg. given intravenously every two hours during operation, is essential. In addition, a sympathomimetic amine such as isoproterenol should be given in doses of 10 mg. sublingually two hours preoperatively and by intravenous drip in a dilution of 4 mg. per liter during the operation. Cardiac activity should be monitored continuously and a cardiac pacemaker-defibrillator unit held in readiness.

An unexplained tachycardia in the elderly patient is a cause of great concern. Apprehension is not the most common cause of this aberration in the preanesthetic period. The operation should be delayed until the cause is determined and appropriate therapy administered. Previously unsuspected organic heart disease or hypovolemia is usually found to be responsible. The danger of tachycardia lies in the shortening of the diastolic phase of the cardiac cycle, which may precipitate coronary insufficiency or myocardial decompensation.

With advancing age the heart becomes less sensitive to the effects of atropine and more sensitive to carotid sinus stimulation. This increased sensitivity of the carotid sinus may be an important cause of sudden cardiovascular collapse during surgery. It may be enhanced by inflammation in the anterior triangle of the neck from infection or radiation therapy; by such drugs as digitalis, morphine, thiopental, cyclopropane, and antihypertensive agents; and possibly by hypoventilation associated with hypoxia and hypercarbia. Stimulation of the carotid sinus may result during operation from extension of the neck or from manipulation during a radical neck dissection. Marked hypotension associated with bradycardia will ensue, and appropriate doses of atropine sulfate should be administered intravenously[6].

The risk involved in administering anesthesia to the patient with aortic valvular disease is particularly grave. These patients are subject to sudden death from coronary occlusion and ventricular fibrillation.

Pulmonary Function

The two main functions of respiration are the intake of oxygen and the elimination of carbon dioxide. Oxygen intake is dependent on the integrity of both ventilation and diffusion. However, carbon dioxide diffuses 20 times more readily than oxygen and depends almost entirely on ventilation for its successful elimination.

Chronic bronchopulmonary disease is a frequent accompaniment of old age. When defects in pulmonary diffusion and ventilation are present, the superimposed stress of anesthesia and surgery may produce serious metabolic and electrolyte disturbances. Respiratory acidosis associated with hyperkalemia will reduce the effectiveness of digitalis therapy and produce alterations in cardiac function. The vital capacity, maximum breathing capacity, and inspiratory reserve volume decrease with advancing age. There is also a decrease in the area of pulmonary capillary—pulmonary alveolar contact, and diffusion studies have shown a more uneven distribution of gases in the lungs. Patients with chronic bronchopulmonary disease may be helped greatly by increasing ventilatory capacity in the preoperative period. This can be accomplished by the use of postural drainage, respiratory exercises, and intermittent positive-pressure breathing with bronochodilator nebulization and detergent aerosols. Intermittent positive-pressure breathing will frequently decrease atelectatic areas and improve ventilatory capacity.

Metabolic Disturbances

Diabetes mellitus is frequently encountered in the elderly patient. Mild hyperglycemia with glycosuria is a relatively harmless condition. Metabolic acidosis, however, is deleterious under any circumstances. The combination of hyponatremia associated with hyperglycemia and hyperkalemia associated with acidosis will produce the characteristic sodium-potassium shift of diabetic acidosis. The elderly diabetic patient usually has severe atherosclerotic vascular disease with decreased renal function. The ability to compensate for metabolic acidosis may be impaired. It should also be emphasized that rapid treatment of diabetic acidosis with insulin may produce hypokalemia.

Careful evaluation and preparation of the elderly diabetic patient for surgery is essential. During operation it is necessary to administer adequate doses of insulin and glucose in order to maintain carbohydrate metabolism without producing hypoglycemia. The risk of hypoglycemia is considerably increased by the administration of drugs which block the sympathetic nervous system, since they inhibit glycogenolysis by epinephrine. The normal symptoms of a hypoglycemic reaction are due in part to the reflex sympathoadrenal release. Sympathetic blockade may mask the symptoms of hypoglycemia.

Summary

Careful attention must be given to the preanesthetic evaluation and preparation of the geriatric patient. The detrimental aspects of inadequate preparation are particularly impressive in this age group. There are few emergency situations in which time cannot be taken to institute measures which will improve the patient's chances of survival. In general, most older patients will tolerate anesthesia well, provided they are suitably prepared and the anesthesia is properly managed.

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Premedication in the Geriatric Patient

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CONCORD

It is generally assumed that a patient enters the geriatric age sometime during his sixth decade. It is important to remember that chronologic age is not an accurate guide to physiologic age. Many 60 year old patients appear to be much older than some who are 70. This fact is important in the choice of premedication, because the patient's physiologic age has a far greater influence on his response to these drugs than does his chronologic age. His physiologic age "is determined by the functional capacity of his various organs, his mental capacity, his appearance and his responses to stress and strain."[1] An understanding of the physiologic changes that occur in the aged is essential, therefore, to proper medication in this age group.

Factors Affecting the Choice of Drugs

The geriatric patient has a lower metabolic rate than does a younger person[2], and his renal and hepatic function is often decreased as a result of degenerative changes. Consequently he is less able to detoxify and eliminate drugs. His reflex excitability is also decreased, which means that he requires a smaller amount of a depressant for adequate premedication than does a younger person.

Since effective premedicant drugs tend to depress the central nervous system and the cardiovascular, respiratory and renal systems, even in the young healthy patient, extreme care should be taken to avoid overdepression in a geriatric patient who has either impaired reserve or actual impairment of function of these various organ systems. What may be considered normal premedicant dosage in a young patient may be gross overdosage in an elderly one.

One of the most likely and most serious effects of overdosage in premedication is respiratory depression producing hypoxia and hypercarbia. Equally hazardous is the production of hypotension from depression of the sympathetic nervous system, and from the hypoxia due to the respiratory depression. A patient so depressed may enter a vicious cycle requiring resuscitative efforts to prevent death.

The purpose of premedication in geriatric patients is much the same as in anyone else. Probably the most desired effect is psychic sedation, in order that the patient may be calm and somewhat indifferent to his surroundings when he arrives in the operating room. Secondly, it is important to minimize secretion of mucus in the respiratory tract, although with the popularity of anesthetics which are parasympathetic in action, the problem of secretions is not as great as with ether. Other purposes of premedication are to decrease reflex activity, to diminish metabolic activity, and to fortify certain anesthetics[3].

The basic concept of premedication has not significantly changed over the years, although the methods and the types of drugs used are varied constantly. With the multiplicity of premedicant drugs now available and the constant search for new ones, it is rather obvious that there is no universal ideal drug.

Morphine has been given to patients prior to the induction of anesthesia since 1850[4]—virtually since ether anesthesia was discovered. It is used widely today and is still a good drug for this purpose, but like many drugs it has adverse side effects. Therefore, the search for a better premedicant has continued. A wide range of drugs is now available for use in premedication, including narcotics, barbiturates, ataractics, tranquilizers, and antiemetics. Since these all have undesirable side effects, and since there is some difference of opinion concerning what constitutes ideal preanesthetic medication, it behooves the anesthetist to beware of any new drug which is said to promise more than drugs well proven by years of successful experience.

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In general, basic premedication should consist of a sedative and an antisecretory drug administered approximately one hour prior to the induction of anesthesia. Other types of drugs may be safely and effectively added according to the physician's experience and knowledge.

Narcotics vs. Barbiturates

There has been considerable controversy concerning the use of narcotics versus barbiturates for sedation. Some authorities recommend withholding narcotics unless the patient is in pain[4]. They prefer barbiturates on the grounds that the latter cause less respiratory depression and spare the patient the mental depression that follows the euphoria produced by morphine. Adriani, however, challenges the routine substitution of barbiturates for narcotics on the basis that enough barbiturate to produce adequate sedation is likewise enough to depress the respiration, and is still not as effective in relieving anxiety[3].

Jacoby, of Marquette University, advocates combining a narcotic with a barbiturate. Meperidine (Demerol) produces less respiratory depression than does Morphine[5]. Since Meperidine produces less euphoria, a barbiturate (Nembutal or Seconal) is added to increase the patient's indifference to his surroundings. My practice is to administer the barbiturate at least two hours and the narcotic one hour prior to the induction of anesthesia.

In general, barbiturates are poorly tolerated by the geriatric patient. They frequently produce marked respiratory or circulatory depression before, during, or after anesthesia[1]. They may also produce some degree of confusion and lack of cooperation. It may therefore be advisable to substitute a drug such as chloral hydrate or an ataractic.

Ataractics

Recently there has been much enthusiasm for tranquilizers, or ataractics, as adjuncts to premedication. It is the choice between these agents that seems to be causing the most confusion at present.

The word "ataractic" is derived from a Greek word meaning "peace of mind"[6]. In contrast to the narcotics and sedatives, which act on the cerebral cortex, the ataractic drugs appear to act primarily on the subcortical structures concerned with the control of emotions[3]. They have the ability to calm the hyperactive, agitated individual without causing marked confusion or drowsiness. This effect may be demonstrated experimentally in animals. An animal under the influence of an ataractic eats normally and does not appear to sleep more, but his physical and emotional responses to a threatening situation are changed—that is, he may show indifference to an attack by another animal where previously he would have exhibited fright[6].

The effectiveness of ataractic drugs in emotionally disturbed patients has been well established, but their effect on normal individuals is less certain. When used alone they are weak and ineffective for preoperative sedation, and some of them produce varying degrees of ganglionic blockade which may lead to an unexpected state of hypotension. In view of these considerations, they should be used with caution and discrimination.

Beckman[6] has divided these agents into four principal groups accroding to their chemical structures:

1. *Rauwolfia derivatives*
    - a. Rauwolfia (Raudixin, Rauserpa)
    - b. Alseroxylon (Rauwiloid)
    - c. Deserpidine (Harmonyl)
    - d. Rescinnamine (Moderil)
    - e. Syrosingopine (Singoserp)

Rauwolfia derivatives were the pioneers among the tranquilizing agents. They have been superceded by the phenothiazine group of drugs, which are more effective ataractics, with a more rapid onset and shorter duration of action.)

2. *Phenothiazine derivatives*
    - a. Chlorpromazine (Thorazine)
    - b. Mepazine (Pacatal)
    - c. Promazine (Sparine)
    - d. Prochlorperazine (Compazine)
    - e. Perphenazine (Trilafon)
    - f. Thiopropazate (Dartal)
    - g. Triflupromazine (Vesprin)
    - h. Trifluoperazine (Stelazine)

    i. Thioridazine (Mellaril)
    j. Methoxypromazine (Tentone)

Attention was drawn to this group of drugs through observations that promethazine (Phenergan), an antihistaminic, also exerts a sedative action. Chemical modifications of this agent led to the discovery of the first of the genuine tranquilizers of this group—chlorpromazine (Thorazine).

3. *Propanediol derivatives*
    a. Meprobamate (Miltown, Meprospan, Meprotabs, Equanil)
    b. Phenaglycodol (Ultran)
4. *Diphenylmethane derivatives*
    a. Azacyclonol (Frenquel)
    b. Hydroxyzine (Atarax, Vistaril)
    c. Benactyzine (Suavitil, Phobex)
    d. Pipethanate (Sycotrol)

Since there are no ataractics that will supplant the hypnotic and the narcotic drugs commonly used, they should, in any event, be used only as a supplement in premedication.

Among the ataractic drugs, promethazine (Phenergan) is highly recommended as a safe, effective agent that has withstood the test of rather wide clinical experience. Its hypotensive effect is minimal. It has both a sedative and a tranquilizing effect, and it appears to enhance the action of narcotics, thereby decreasing the dosage requirements of the latter[3].

Antiemetics and Antisecretory Drugs

The antiemetics comprise another group of drugs that is receiving considerable attention with respect to premedication. These include numerous anticholinergics, antihistaminics, and phenothiazines that exert varying degrees of antiemetic activity. Since many of the antiemetics are far from innocuous and may produce serious side effects, and since it has not been proved that all patients should receive an antiemetic before operation[7], their routine use appears inadvisable in the geriatric patient.

As to the antisecretory or "drying agent" in the premedication, atropine or scopolamine still remain choice agents. Synthetic anticholinergic drugs, oxyphenonium (Antrenyl), methantheline (Banthine), and methscopolamine bromide (Pamine), are less effective and less desirable than is either scopolamine or atropine[3]. Although atropine does not have as great an antisecretory effect as scopolamine, it is preferable in the geriatric patient, in whom scopolamine may at times produce delirium[1].

Summary and Conclusions

Preanesthetic medication for the aged does not differ radically from that normally administered to other adults. The differences are that smaller amounts of the drugs are required, that atropine is preferred to scopolamine, and that the use of barbiturates should be avoided. The chief precautions are the avoidance of respiratory and circulatory depression while striving at the same time to allay apprehension and to decrease salivation.

Elderly patients have less tolerance to depressant drugs than do younger ones. It is recommended that the dose of a narcotic be reduced to one-half, one-fourth, or even one-sixth that required for middle-aged patients[3]. Some very ill geriatric patients may not need any depressant, only an antisecretory drug.

A reliable, safe program of premedication in the geriatric patient is 25 to 50 mg. of promethazine (Phenergan) given intramuscularly two hours prior to induction of anesthesia, followed by 25 to 50 mg. of Demerol with 0.3 mg. of atropine given intramuscularly one hour prior to induction. It may be necessary to administer even smaller doses in debilitated patients. It is better to err in the direction of too little than too much premedication.

In the geriatric patient it is even more important than in the younger patient to individualize the type and the amount of drug to be used.

The basic concept of premedication of the surgical patient has not significantly changed over the years. Although numerous drugs are now available, there is no single universal, ideal drug.

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Anesthetic Agents and Methods for the Geriatric Patient

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The purpose of this paper is to consider the following questions: (1) How does the geriatric patient differ from other candidates for anesthesia and surgery? (2) How do these differences affect the choice of anesthetic agents and methods?

Common Problems in the Geriatric Patient

In characterizing the geriatric patient, one might go back to the words of the man who suggested the name "anesthesia"—Oliver Wendell Holmes. The geriatric patient could very well be likened to his description of "The One Hoss Shay," which, at the end of one hundred years of service, showed

> A general flavor of mild decay
> But nothing local, as one may say,
>
> It went to pieces all at once,
> All at once, and nothing first,
> Just as bubbles do when they burst.

The elderly patient has lost the resilience that is seen in the robust and healthy person of younger years. This loss is most evident in the cardiovascular, respiratory, and genitourinary systems.

Respiratory system

In the respiratory system pulmonary fibrosis, emphysema, and bronchitis are commonly encountered. There is poor mixing of gases, because of regional alterations of the ventilation-perfusion ratio[1] and a resultant drop in arterial pO_2 and an elevated pCO_2. Hyperventilation does not completely correct the hypoxia in this situation, but it can correct the hypercapnia. Regional anesthesia should be utilized when the scope of the surgical procedure permits. Low spinal anesthesia serves very well, in contrast to high spinal anesthesia, which produces profound alterations in cardiac output, peripheral blood flow, and hepatic, renal and cerebral circulation. Respiratory efficiency is further diminished by high spinal anesthesia[2]. The skill of the administrator is an important factor, as evidenced by the disastrous effects of pneumothorax complicating a brachial block in a respiratory cripple.

In the upper airway, reflexes are less active and aspiration is more likely to occur. Obstruction of the upper airway on expiration is frequently a problem in the edentulous patient during induction.

Cardiovascular system

Frequent problems involving the cardiovascular system are arteriosclerosis, coronary insufficiency, cardiac decompensation, and hypertensive cardiovascular disease. Avoidance of hypoxia and hypotension is the common denominator in preventing myocardial hypoxia. Assessment of respiratory function is often the most important consideration in the cardiac patient. An adequate blood volume is essential in preventing hypotensive episodes.

With well managed anesthesia, the stress of surgery may be less than that of the postoperative period, in view of the lowered metabolism and the oxygen-enriched atmosphere during operation. Not infrequently, arrhythmias, particularly premature ventricular beats, will disappear in the monitored, anesthetized, and well ventilated patient, only to reappear in the recovery period.

Hypertension without complications presents little difficulty as long as hypotension is prevented during the anesthetic period. In patients who have been on Rauwolfia alkaloids, operation should be deferred for 10 days or regional anesethsia should be used. Burn and Rand[3] have demonstrated that reserpine depletes norepinephrine from the arterial walls as well as from the hypothalmus, and this factor appears to account for the hypotension that sometimes occurs in these patients. Agents which elicit a sympatho-adrenal discharge may become quite depressant in the catecholamine-depleted patient.

When cardiomegaly is present, a portion of the latent cardiac reserve has been drawn into action. When one recalls that cardiac reserve is the product of three adaptive mechanisms — (1) increase in heart rate, (2) increase in stroke volume, and (3) hypertrophy—it is obvious that less reserve remains to counter any added stress[4].

Congestive failure should be corrected preoperatively by long-acting digitalis preparations, if at all possible. The patient who has been subjected to rapid digitalization is a poor risk in view of the fact that momentary acid-base balance, electrolyte concentration, available energy, and calcium-magnesium-potassium relationships profoundly affect the myocardial effects of digitalis[5]. Hypoxia increases digitalis toxicity, and these patients should be monitored during anesthesia for the appearance of ventricular premature beats, bigemini, atrioventricular block, and paroxysmal atrial tachycardia.

Renal system

Renal insufficiency and obstructive uropathy must be viewed with three factors in mind. First, fluid and electrolyte balance should be corrected as nearly as possible, and urinary drainage established. Second, nonvolatile narcotics and analgesics, especially those depending on renal excretion, must be kept to a minimum. Pentothal, phenobarbital, codeine, gallamine, and decamethonium should be avoided in the presence of renal insufficiency[6]. Third, renal function is depressed by most types of general anesthesia in a rather stereotyped pattern[7]. Morris[8] has reported, however, that light anesthesia with liberal use of muscle relaxants permits nearly normal renal function.

Time should be taken preoperatively to correct any metabolic disturbances, malnutrition, and avitaminosis that may be present. Blood volume deficits are replaced with much greater safety when administered slowly in the conscious patient.

Choice of Agent and Method

In choosing the agents or techniques to be employed, one should strive for (1) safe anesthesia, and (2) optimal operating conditions for the surgeon, in that order[9]. The armamentarium of agents and techniques available for the geriatric patient actually is no more limited than that for other age groups, but the proper choice is much more important in view of his reduced reserve and loss of resiliency. Knight[10] has adroitly stated the situation as follows:

> ---any anesthetic or method, or combination of anesthetics and methods, that is serviceable and good at all is serviceable and good for old people. The main difference is that these patients are tender, are quieted easily and overdosed readily. What they require particularly is conservatism with a little daring mixed in, but with unceasing vigilance and nicety and exactness of administration and control.

How is the "proper choice" made? It is arrived at after a thorough and searching individualized assessment of the patient[9]. It is a kindness to the older patient to defer anesthesia and operation until he has been thoroughly evaluated and adequately prepared. Even under these circumstances it is a fallacy for the anesthetist to assume that the patient has been converted into a "normal risk" by means of the best preoperative management.

The first goal, safe anesthesia, is that which produces the least metabolic disturbance[9]. Regional anesthesia, when skillfully administered and carefully managed, interferes less with metabolic functions than does general anesthesia, and should be considered as the technique of choice when the extent and site of the operation permits. For a number of reasons the use of regional anesthesia has declined during the past two decades. Some of the reasons are valid—such as the improvement in general anesthetic agents and techniques, the better understanding of the physiologic effects, and in particular, the advent and proper use of muscle relaxants.

Other reasons for opposing a regional approach bear further discussion. Bonica[11] has listed these objections as follows: (1) Patients do not like the method; (2) it is time-consuming; (3) there are too many failures; (4) serious complications can occur; (5) the medicolegal risk is greater; and (6) most surgeons dislike the method.

Aversion to regional anesthesia is uncommon in the geriatric patient, who has been found to be much more compliant than the young, robust person. The use of reason and reassurance during an unhurried preanesthesia visit, and good results with the management, dispel any dislike of the procedure even in the younger patient.

Most regional techniques take little or no longer than does a properly administered general anesthetic, and even if they did, it is not morally right to compromise a patient's welfare for expediency. Failures, or faulty administration, occur with general as well as with regional methods. The regular use of regional techniques and lack of hesitancy in repeating or adding to the block precludes any significant number of failures. With a cooperative surgeon, supplementation by local injections is another logical approach. For the block that begins to "wear off," supplementation is easily obtained with the judicious use of analgesics and hypnotics.

In experienced hands, complications are no greater a problem with regional than with general anesthesia. In the past, law suits over complications following, but not necessarily due to, subarachnoid block have caused many anesthesiologists to allow medicolegal considerations to supersede the patient's welfare. Dislike of regional anesthesia by the surgeon is observed in areas where it is seldom utilized. Surgeons who have been exposed to well conducted regional anesthesia will frequently request it in preference to general. Regional anesthesia is not a panacea, but it should be employed when there is a clear-cut indication.

When general anesthesia is utilized, it must be remembered again that the mark of safe anesthesia is minimal disturbance of metabolic functions[9]. In the geriatric patient this can be achieved only by employing light planes of anesthesia and minimal quantities of hypnotics and analgesics; by avoiding hypoxic gas mixtures; and, most important, by assuring adequate pulmonary ventilation. With the advent of the muscle relaxants, it is now much easier to provide satisfactory operating conditions while still meeting the above prerequisites for safe anesthesia in the geriatric patient.

Summary

The geriatric patient represents an increased risk for anesthesia and surgery, owing to his diminished resilience, particularly in the cardiovascular, respiratory, and genitourinary systems. The choice of anesthetic agents and methods for the older patient is based upon individualized preoperative assessment and preparation.

Regional anesthesia should be more widely employed in cases where it is indicated, if we are to have a more complete armamentarium to circumvent metabolic disturbances. If general anesthesia is utilized it must be kept in light planes, with particular attention paid to adequate pulmonary ventilation and avoidance of hypotension.

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Postanesthetic Care of the Geriatric Patient

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The geriatric patient is similar to the pediatric patient in many respects. His reflex irritability is decreased, his tolerance narrowed, and his response to stress diminished. Unlike the infant, however, the senior citizen is subject to a bevy of aging processes which harden his arteries, weaken his brain, and limit his pulmonary reserve. As his requirement for anesthetics decreases, so does his resistance to insults, and nowhere is this process more apparent than in the immediate postanesthetic period. It behooves all attending personnel, therefore, to give the elderly patient the meticulous care he needs and deserves.

The postanesthetic care of the geriatric patient, as with all patients, begins with the preanesthetic evaluation. Much postanesthetic morbidity and mortality can be avoided by careful preanesthetic and peranesthetic management. A brief outline of the postanesthetic care by review of systems, with emphasis on the cardiovascular and respiratory systems, will be presented here.

Cardiovascular System

The function of the cardiovascular system is to maintain an effective circulating blood volume with its concomitant transport of nutrients and oxygen to the cells, and carbon dioxide and other waste products away from the cells. When it fails to do this, a syndrome called "shock" ensues. The system has three main components: the blood volume, the heart, and the peripheral vasculature. All three components are likely to be impaired in the geriatric patient; therefore, the margin of safety is narrow, as it is in the pediatric patient.

First, the blood volume is likely to be low in the geriatric patient because of inactivity, poor nutrition, and other factors. After surgery it is likely to be even lower, unless blood loss was meticulously replaced during the anesthetic period. Even if his blood volume is low, the geriatric patient probably is well adjusted to it, and be cannot tolerate sudden changes in either direction[1]. Rapid blood loss can quickly cause peripheral vascular collapse, whereas too rapid transfusion can cause circulatory overload leading to heart failure and pulmonary edema. It is frightening to be caught between circulatory overload on the one hand and peripheral vascular collapse on the other, an easy trap to fall into with the geriatric patient.

Second, the heart can fail in the postanesthetic period for a variety reasons.

1. *Preexisting heart disease*. In the older patient this will usually be of a degenerative nature, such as arteriosclerosis, rather than of congenital origin, such as the valvular defects of the younger patient. Functionally, cardiac impairment may manifest itself in two ways: as a decrease in *stroke volume* because of weakening of the myocardial contractility; or as a decrease in *heart rate* because of a defect in the conduction system, with a resultant arrhythmia which will essentially decrease the number of effective beats per minute. A decrease in either the stroke volume or the heart rate may cause a decrease in the effective circulating blood volume.

2. *Anesthetic agents*. All general anesthetic agents depress the myocardium directly, roughly proportional to their concentration in the blood. Some of them may interfere with the conduction system of the heart,

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particularly when combined with substances such as epinephrine. These effects, therefore, reinforce the deleterious effects of any preexisting disease as described above. It is a mistake to think that these effects do not persist into the postanesthetic period. Considerable amounts of all agents used can be found in the circulating blood long after a patient is "awake" from the anesthesia.

3. *Overload or underload of the blood volume.*

4. *Hypoxia, alone or in combination with any of the above factors.* Since chronic hypoxia can be very insidious in the geriatric patient, this is probably one of the greatest contributing factors in heart failure.

5 *Hypercarbia,* particularly sudden shifts in the pH and potassium balance, can precipitate severe arrhythmias.

6. *Other factors such as cold and electrolyte imbalances*—for example, rapid transfusion of citrated blood causing sudden changes in the calcium-potassium ratio.

Third, some peripheral resistance is necessary to maintain an effective circulating blood volume, particularly to compensate for positional changes (gravity forces) and to shunt blood from one organ system to another. The geriatric patient usually has some degree of sclerosis and loss of tone in the peripheral vessels, which makes it difficult for him to adjust to these changes rapidly. If he is moved about suddenly during the immediate postanesthetic period, severe drops in blood pressure are likely. All the factors that adversely effect the myocardium may also impair the peripheral circulatory bed, such as hypoxia, electrolyte imbalance, cold, and anesthetic agents. The control of peripheral resistance is maintained largely on the arterial side. The venous circulation is important also, in that it returns blood to the heart. A stagnant venous circulation can cause severe difficulty in the geriatric patient. Much of this return depends upon muscle tone, and activity, both of which may be severely limited in the older patient. Thrombosis of the major veins with resultant stagnation, emboli, or both can be fatal sequelae in the postanesthetic period.

Management

How can one deal with these cardiovascular problems in the geriatric patient? First, the blood volume must be carefully evaluated in the postanesthetic period and any deficiencies or excesses corrected. In this regard it must also be remembered that some patients will continue to bleed in the postanesthetic period. All drainage containing blood from chest suctions, dressings, and other sources should be measured and the estimated loss replaced. Suspect a low blood volume in any patient who does not wake quickly from anesthesia or whose vital signs are unstable.

Because of the depressant effect of persisting anesthetics, one must not transfuse too rapidly and overload the circulation before the heart and kidneys can handle the extra volume. A simple and effective tool to help one steer between the Scylla of overload and the Charybdis of collapse is to monitor the venous pressure as the patient is being transfused. Careful titration of arterial and venous pressures, during transfusion will rarely lead to overload.

Second, once the blood volume is stabilized as well as possible, any causes of primary heart failure must be corrected. Good ventilation and oxygenation are essential to the geriatric patient. Electrolytes must be balanced. All anesthetics must be removed as quickly as possible. The "stir-up routine"—to be discussed later—will help speed up the elimination of the inhalant agents.

After these matters have been attended to, digitalization must be considered. Many elderly patients will already be digitalized. Since most anesthetic agents potentiate the effects of digitalis, underigitalization is rarely a problem in the immediate postanesthetic period. If such signs as tachycardia and increased venous pressure suggest failure, the dosage may have to be increased. One should be very cautious while anesthetics persist in the patient's circulation, however, and underdosage is probably safer than overdosage during the immediate postanesthetic period. The same holds true for the nondigitalized patient in whom digitalization is being considered.

Cardiac stimulants should be used with

great caution, but if a heart is failing and all other measures have been tried, such drugs as calcium and isoproterenol (Isuprel) are occasionally useful. Calcium is of particular value when multiple transfusions have been given and the possibility of a lowered calcium-potassium ratio exists. Isuprel, of course, is most useful in patients with an atrioventricular block, but sometimes will also improve myocardial tone, *per se*. Atropine-like drugs are useful for severe bradycardia, although many geriatric patients have a "fixed vagal tone" and show little response to the anticholinergics.

Third, because of the poor control that geriatric patients have over their peripheral resistance, they must be moved very gently. Legs should be lifted slowly out of lithotomy stirrups, other positional changes made carefully, and the transition from stretcher to bed made smoothly. Sometimes a small dose of a mild vasopressor such as methoxymine (Vasoxyl) or metamphetamine (Methedrine) is helpful in maintaining normal peripheral resistance during this transitional stage. Vasopressors should *never* be used to reverse hypotension due to deep anesthesia, hypoxia, or low blood volume except in one circumstance. If life is believed to depend on maintaining a high arterial pressure in order to perfuse the coronary or cerebral arteries, and if all other measures fail, it may be necessary to try vasopressors. If this is done, however, the primary defect should be corrected and the vasopressor withdrawn as soon as possible. With sympathetic blockade, such as in spinal anesthesia, mild vasopressors *are* indicated to maintain peripheral resistance until the blockade ends.

Venous stagnation can best be prevented by frequent active and passive exercise. The best position for the postanesthetic geriatric patient is a changing one. Intravenous drips should be discontinued as soon as possible. If prolonged fluid therapy is necessary, the intravenous needle should be rotated to different veins.

Metabolic System

While it is not the purpose of this discussion to detail the many metabolic disorders that the geriatric patient is susceptible to, it should be noted that they are many and that the immediate postoperative period is one of the most crucial in this regard. These patients should be carefully regulated. Even though they may have been in reasonable metabolic control before, the stress of anesthesia and surgery will often seriously disrupt this balance.

Diabetic patients may increase their demand for insulin threefold or more, or they may suddenly become hypoglycemic. A relatively fast intravenous glucose drip should be maintained in the postanesthetic period until they are able to retain oral fluids. The urine should be examined for sugar and acetone as soon as possible. If the patient has not voided in two hours, he should either be catheterized or have a blood sugar determination, particularly if he has not regained a good level of consciousness. It is far better for him to be spilling some sugar, as long as the acidosis is controlled, than to become hypoglycemic. Only regular insulin should be used.

Patients with severe liver disease must be closely watched. The blood sugar level should be well maintained, and they should, of course, be kept well oxygenated. "Cocktails" containing massive doses of thiamine, some insulin, and glucose may be helpful. If the patient is a severe alcoholic and shows a tendency to have delirium tremens, he should be given alcohol immediately—orally, if tolerated; intravenously, if not—in about a 5 per solution.

The fluid balance of patients with severe renal impairment must be carefully controlled. Urinary output must be watched via an indwelling urethral catheter, and fluids should be limited until output is satisfactory. Lost protein must be replaced. Hypotension should be avoided. Vasopressors must be used with extreme caution, however, as they may actually decrease blood flow.

One factor that must be considered in all geriatric patients, particularly those with metabolic diseases, is hypoadrenalism. This condition may be precipitated by the stress of anesthesia and operation in an elderly patient, but is more common in (1) those who have previously taken steroid preparations and who were not given booster doses before anesthesia; (2) those with diseases, such as tuberculosis, which may have destroyed functioning adrenal tissue; (3) those who have been under stress, as from a long illness; and (4) those in severe states of malnutrition.

If circulatory collapse occurs after anesthesia in any geriatric patient, and if all other avenues have been explored, a course of steroids is well worth trying. Cortisone hemisuccinate, given intravenously, will produce the most rapid response (15 to 20 minutes), but will not maintain a blood level unless it is also added to a continuous drip. Since it is almost impossible to give overdoses of these preparations, or give them too rapidly, massive doses should be tried in an acute situation. At least 200 mg. given intravenously in a single shot to start with, and 200-400 mg. in the drip should be tried. After a satisfactory response has been attained, the patient may be given 100 mg. of cortisone acetate twice daily, after which the drug may be tapered off over at least a three-day period.

Respiratory System

Three major pulmonary problems beset the geriatric patient in the postanesthetic period: pulmonary circulatory impairment, excessive secretions, and deficient aeration of the alveoli. Circulatory impairment has been discussed in relation to the cardiovascular system.

Both excessive secretions and deficient alveolar aeration may be caused by a variety of factors, and both are of considerable importance in any elderly patient. Those undergoing lung surgery, however, present an especially serious problem with regard to these two conditions.

Secretions may be increased by (1) preexisting pulmonary disease, such as tuberculosis, fungus infection, bacterial pneumonia, or bronchial asthma; (2) accumulation from lack of gross body and pulmonary movement, coupled with a markedly decreased cough reflex, so common in elderly people[2]; (3) certain drugs, particularly some of the general anesthetic agents such as ether; (4) surgical intervention, which may not only stimulate the production of secretions in general, but by manipulation, cause secretions or even pus to move from diseased to healthy areas of the lung. It may also introduce another troublesome substance into the tracheobronchial tree—namely, blood.

Inadequate aeration of the alveoli may also result from many factors:

1. *Impairment of the control of respiration due to damage to the central nervous system.* Degenerative diseases such as arteriosclerosis of cerebral vessels or the so-called "small strokes" that are common in older persons can affect the respiratory centers as well as other parts of the brain.

2. *Mechanical changes in the lung-thorax system.* The older one gets, the less compliant he becomes. Fibrous tissue replaces muscle, and ultimately the "fixed chest" is seen. The patient in this condition relies almost entirely upon his diaphragm for respiratory movements.

3. *Preexisting disease* can reduce alveolar ventilation by collapse and blockage of air passages, and coupled with increased secretions can cause a vicious cycle.

4. *Inactivity* again will decrease ventilation generally or may cause focal atelectasis.

5. *Depressant drugs, including the anesthetics just administered, premedicaments still acting, and postanesthetic analgesics injudiciously given.*

6. *Surgical intervention of the pulmonary system.* Even the most discrete resection of the lung may remove considerable good tissue along with the bad. For this reason, if extensive resection, particularly a pneumonectomy, is anticipated, extremely careful preoperative evaluation of pulmonary function including pulmonary function tests is mandatory[3]. This will help to assess the amount of good functioning lung that may be expected to remain after surgery.

Management

How does one cope with execessive secretions and improper aeration in the postanesthetic geriatric patient? First, a good airway is an obvious prerequisite in the handling of both problems. It must be obtained, and maintained, by whatever means at hand: positioning, holding the jaw, oro- and nasopharyngeal airways, endotracheal intubation, or tracheotomy. Second, every postanesthestic patient must frequently be turned and helped to cough and breathe deeply[4]. This is still the simplest and best method of removing secretions from the tracheobronchial tree and promoting better aeration of the lungs. It should not be a routine order to be written and forgotten, but a vital responsibility that the anesthesiologist must personally see carried out. If the postanesthetic nursing care is not adequate (and so frequently it is not), it behooves the anesthesiologist to administer this care to his patient personally, and to instruct his nursing personnel continually in order to improve their technique. In the geriatric patient, this "stir-up" routine may be necessary every 15 minutes or oftener.

Third, if these measures are inadequate, they must be supplemented with pharyngeal suction to remove secretions and stimulate the cough reflex, endotracheal suction, and tracheotomy, in that order.

Fourth, all postanesthetic geriatric patients should receive some supplementary oxygen, usually by way of a nasal catheter at about 5-7 liters per minute. Although the oxygen content may be increased by only a small percentage, this small boost may be very important in supplementing other therapy aimed at maintaining good arterial oxygen saturation. It must be remembered, of course, that the administration of oxygen will not alter the arterial pCO_2. If this value is high, other therapy such as assisted ventilation must be used. Some will mistakenly criticize the use of oxygen therapy on the basis that it will depress the hypoxic respiratory drive. In an *acute* situation, such as in the postanesthetic period, however, *no* patient should be allowed to depend upon his hypoxic-carotid body drive. If he is that refractory to the normal respiratory drives (principally carbon dioxide), he needs other vigorous treatment.

It is so easy to emphasize good ventilation during anesthesia and then forget about it during the period following, under the mistaken assumption that all the anesthetic has "worn off." This is a very critical time for the elderly patient, a point that leads to the fifth major factor in the postanesthetic care of the geriatric patient—namely, ventilation.

If the patient's ventilation is not adequate as assessed by clinical signs and symptoms, or better yet by arterial pO_2 and pCO_2 determinations, two things may be done. If the impairment is only slight, reduction of the respiratory dead space by a well performed tracheotomy may of itself relieve the acute problem. If it does not, assisted or controlled ventilation by "hand-bagging" or a mechanical respirator is mandatory. Respirators should be used only by those competent to operate them, however, and if there is any doubt, hand-bagging is safer. Even if mechanical ventilation is planned, it is best to stabilize the patient for a few minutes with hand-bagging. This will force the anesthesiologist to pay close attention to the patient and give him the opportunity to adjust the respirator for the most efficient ventilation of that particular patient.

Tracheotomy

The value of a tracheotomy in the postanesthetic geriatric patient, particularly in the one undergoing pulmonary surgery, cannot be overemphasized. There are frequent objections to performing a tracheotomy unless it is "absolutely necessary." These objections usually revolve around the possibility of cosmetic deformity or tracheal stenosis. In the pediatric patient they may be justified, although I do not think so. Stenosis is rare in older people, however, and most of them are more concerned for their comfort and safety than their appearance. Complications occassionally attend tracheotomies, but they are surprisingly few, and the value of the procedure usually so outweighs the risks that the arguments against it are weak.

Tracheotomy provides four major advantages in the postanesthetic care of the older patient:

1. It provides a good airway.
2. It provides an easy access through which even relatively inexperienced personnel may remove tracheobronchial secretions.
3. It materially reduces the respiratory dead space.

4. It provides a controllable access for assisted or controlled respirations. In this event, some type of cuffed tube may be necessary.

Other precautions

A few other factors may be mentioned in passing. Obviously, good chest suction must be provided in any operation that encroaches on the pleural space. The object is to remove air, blood, and other secretions from the pleural space and to keep the lung expanded. Multiple tubes may be necessary to achieve this purpose. The value of the procedure must be remembered in operations less obvious than thoracotomy—for example, explorations of the kidney, mediastinal operations, or low neck procedures. Any patient in the lateral position during operation must be turned in the immediate postanesthetic period in order to promote drainage from the "good" lung and also to promote expansion of this lung, which may have become atelectatic during long periods of positional pressure. After a pneumonectomy either chest suction catheters should be used, or, in the event of a closed chest postoperatively, the intrapleural pressures should be checked frequently with the patient in the supine position and a negative pressure left on the side from which the lung was removed. This keeps the remaining lung expanded and prevents mediastinal shift. Alevolar-capillary block can present a severe problem, particularly in the postanesthetic period, but little can be done about it, *per se,* except to prevent further damage to the alveolar membrane by avoiding hypoxia, secretions, and alveolar collapse.

Finally, the postanethetic geriatric patient must be handled with "tender loving care." He is very likely to acquire pressure sores, sheet burns, and bruises. This can be especially trying in the semi-delirious patient who is thrashing about. He must be restrained firmly but gently, and kept well padded. At the same time, frequent changes in position are mandatory. *An order to turn an elderly patient should be considered as important as an order for an antibiotic or an anticoagulant.* Ice bags and hot water bottles should be used *only* when absolutely necessary, and then should be checked for temperature and frequently rotated to different places on the patient's skin. Good mouth care and general cleanliness is important in these senior citizens, who are frequently unable to care for themselves.

One final word: Frequently the postanesthetic geriatric patient will be disoriented and confused, primarily because of cerebral hypoxia on either a respiratory or circulatory basis. This condition should be treated by improving the airway (with trachetotomy if necessary), administering oxygen, and providing good circulation to the brain. It *should not* be treated with sedatives of any kind.

Conclusion

With good postanesthetic care the geriatric patient will recover quickly and continue to lead a useful life. If these factors are kept in mind, age alone need never be a contraindication to major anesthesia and surgery.

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New Concepts in the Management of Facial Trauma

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Soft tissue injuries of the face are seen with increasing frequency in the physician's daily practice. The ever growing number of automobiles, greater industrialization, and the many farm and home accidents present an abundance of possibilities.

If the principal problem facing the physician is the facial trauma, careful reconstruction of the injured tissues is necessary in order to hold permanent scarring to a minimum. The requisites for an acceptable cosmetic result are careful handling of the tissues, care in removing all foreign bodies possible, and finally closure of the lacerations without tension, utilizing fine suture material. The most able physician is handicapped in handling these injuries if the finer instruments, needles, and newer synthetic suture materials are not available in the emergency and operating rooms of his hospital.

General Principles

1. *Timing.* Facial lacerations should be closed, if the patient's general physical status permits, within the first 24 hours. Because of the excellent blood supply of the facial tissues, coupled with the effectiveness of broad spectrum antibiotics, this is now considered to be the safe period. The usual early use of tetanus toxoid or tetanus antitoxin following skin testing is still important. After this precaution the lacerations can often be closed using a local anesthetic agent such as a 1-2 per cent solution of Xylocaine, with or without epinephrine in a concentration of 1:100,000.

2. *Wound preparation.* Before any move to explore the wound or apply a local blocking agent, the traumatized area and adajacent portion of the face are cleaned. The wound is first carefully probed with swabs saturated with hydrogen peroxide, allowing this to froth and bring up any loose particles and other debris. The surrounding tissues are cleaned with pHisoHex or aqueous Zephiran (1:1,000) with copious amounts of isotonic saline solutions.

3. *Hemostasis.* Prior to any attempts at closing the wounds, it is essential to control any bleeding or oozing. Failure to do this or to clamp the bleeding vessels and tie them with a 4-0 plain catgut suture will result in the formation of hematomas which undoubtedly will become infected and delay healing, with resultant scarring.

4. *Wound exploration.* The early exploration of the traumatized tissues should be practiced in every situation. Lacerations of Stenson's duct, if present, should be repaired immediately in the following manner. An 18-gauge polyethylene tube should be inserted the length of the duct through the oral orifice and across the line of severence. The laceration of the duct is then repaired with interrupted 6-0 sutures. The tube, which is inserted via the mouth and across the lacerated area, is sutured in position for four weeks.

Severed branches of the facial nerve are reapproximated with 6-0 or 7-0 silk on an atraumatic needle. Use of a nerve stimulator makes it easier to locate the severed distal branches of the nerve so that they can be reunited accurately.

Lacerations of the nasolacrimal duct are repaired in the same manner as those of Stenson's duct, being careful, however, to use a smaller tube, usually 20 gauge, which extends through the lacrimal sac and canaliculus as well as into the nose. The tube is kept in position for three to four weeks. Usually a 4-0 chromic catgut suture is used to prevent movement. Care is taken to prevent irritation of the cornea by the tubing.

5. *Traumatic tattooing and abrasions.* Although facial abrasions usually heal satisfactorily if the dermis is largely intact, special attention must be given to removing any foreign particles such as road cinders,

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From the Division of Plastic and Maxillofacial Surgery, Duke University School of Medicine, Durham, North Carolina.

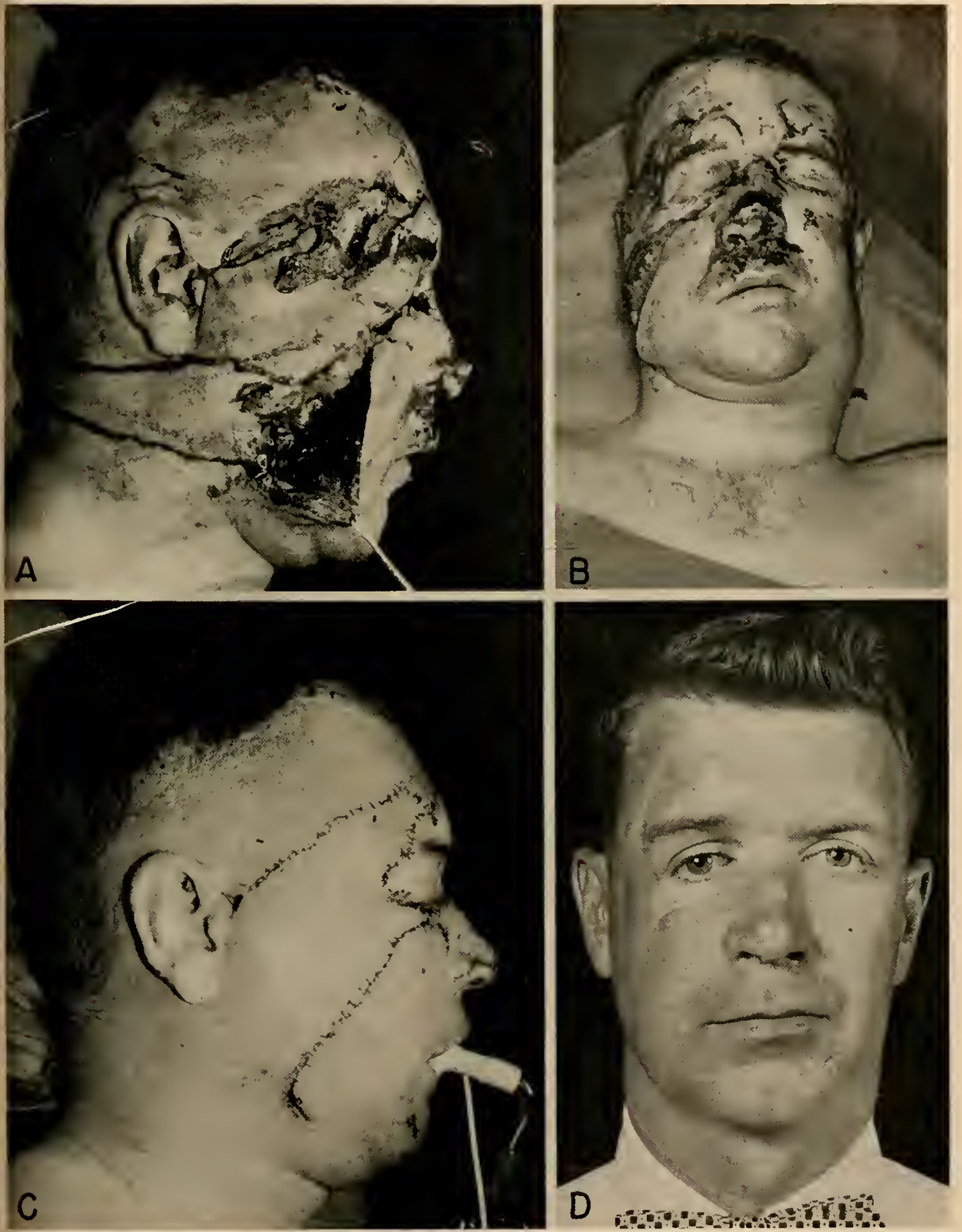


Fig. 1. A and B. Views of facial injuries four hours after automobile accident. C. Lacerations closed in layers utilizing fine synthetic suture material to approximate skin edges. D. Photograph showing results two months after repair.

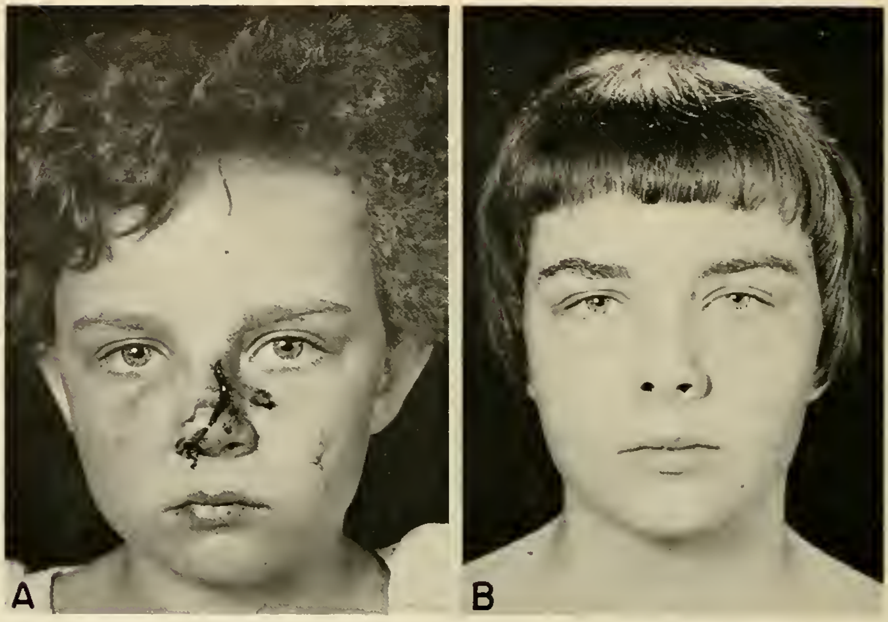


Fig. 2. A. Through-and-through laceration into nasal cavity. B. Photograph taken five years after repair.

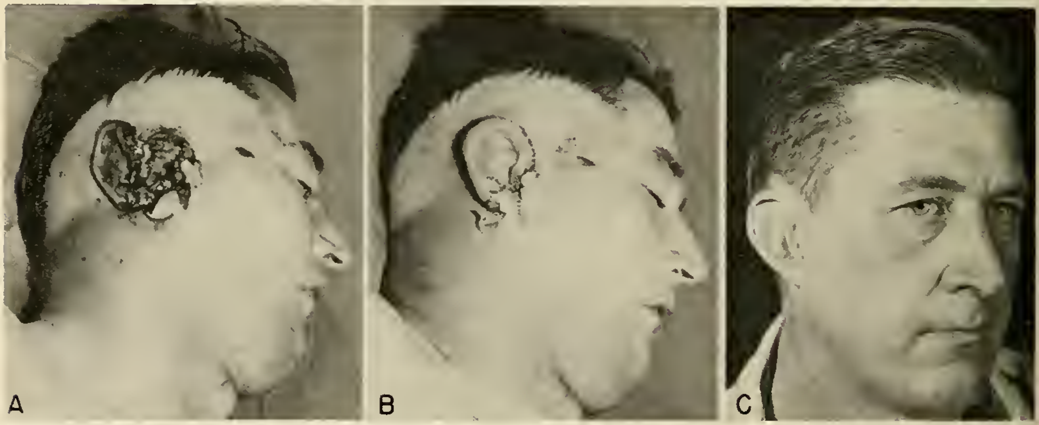


Fig. 3. A. Almost completely severed right ear as seen six hours following accident. B. Repair and immediate reconstruction of ear utilizing multiple small synthetic sutures. C. View of ear three months after repair.

grease, or gunpowder (in the event of a blast injury). The area should be cleaned carefully, using a brush as well as needles if necessary to remove the embedded particles. Otherwise, after healing has occurred they will be visible through the overlying skin and quite disfiguring.

6. *Irregular lacerations and avulsions.* Multiple small, irregular lacerations should be converted to a more regular, easily repaired, and cosmetically acceptable scar by careful excision along the borders. Small areas of avulsion can usually be repaired by primary closure with undermining of the adjacent tissues. Large areas with resultant flaps of tissue must be carefully evalu-

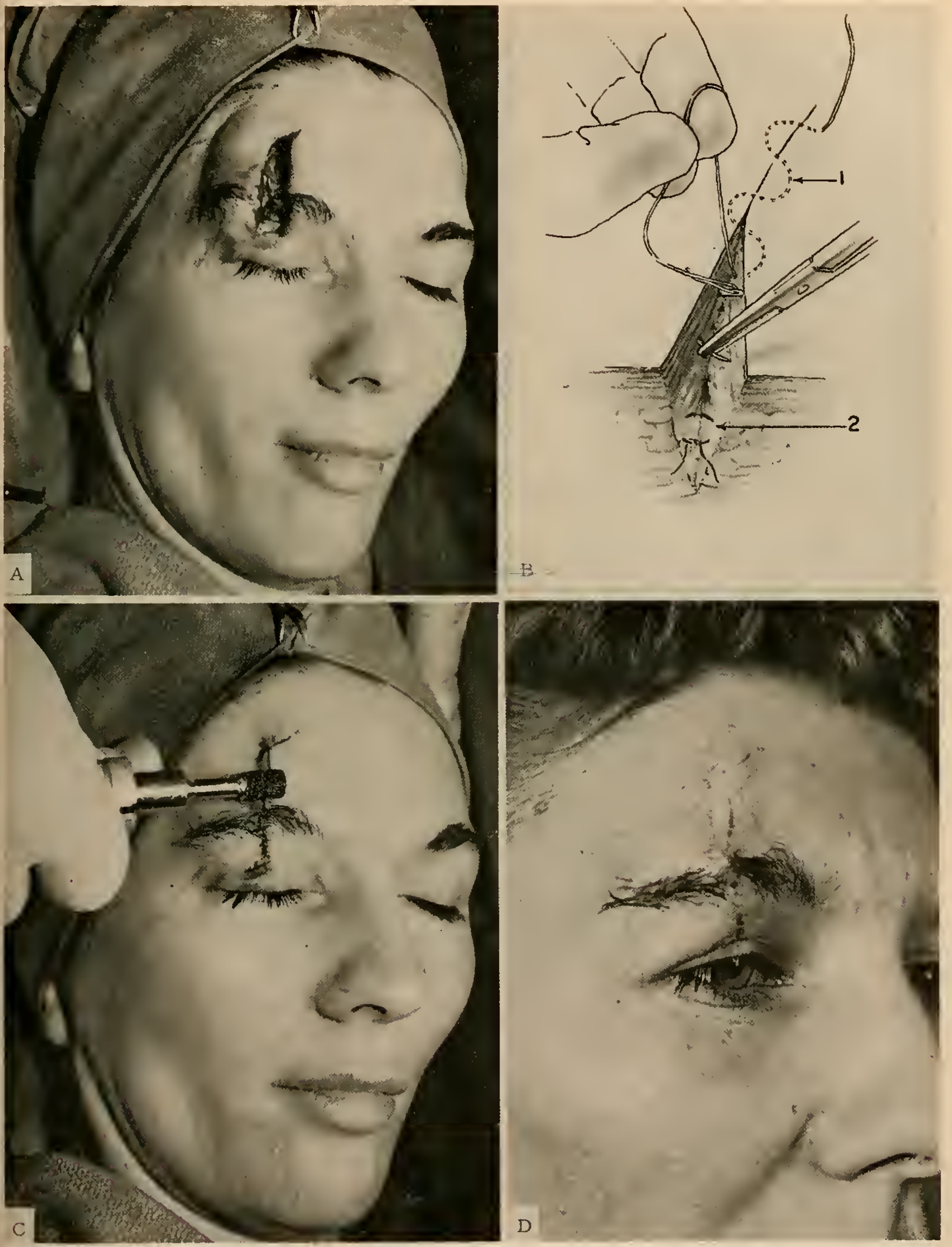
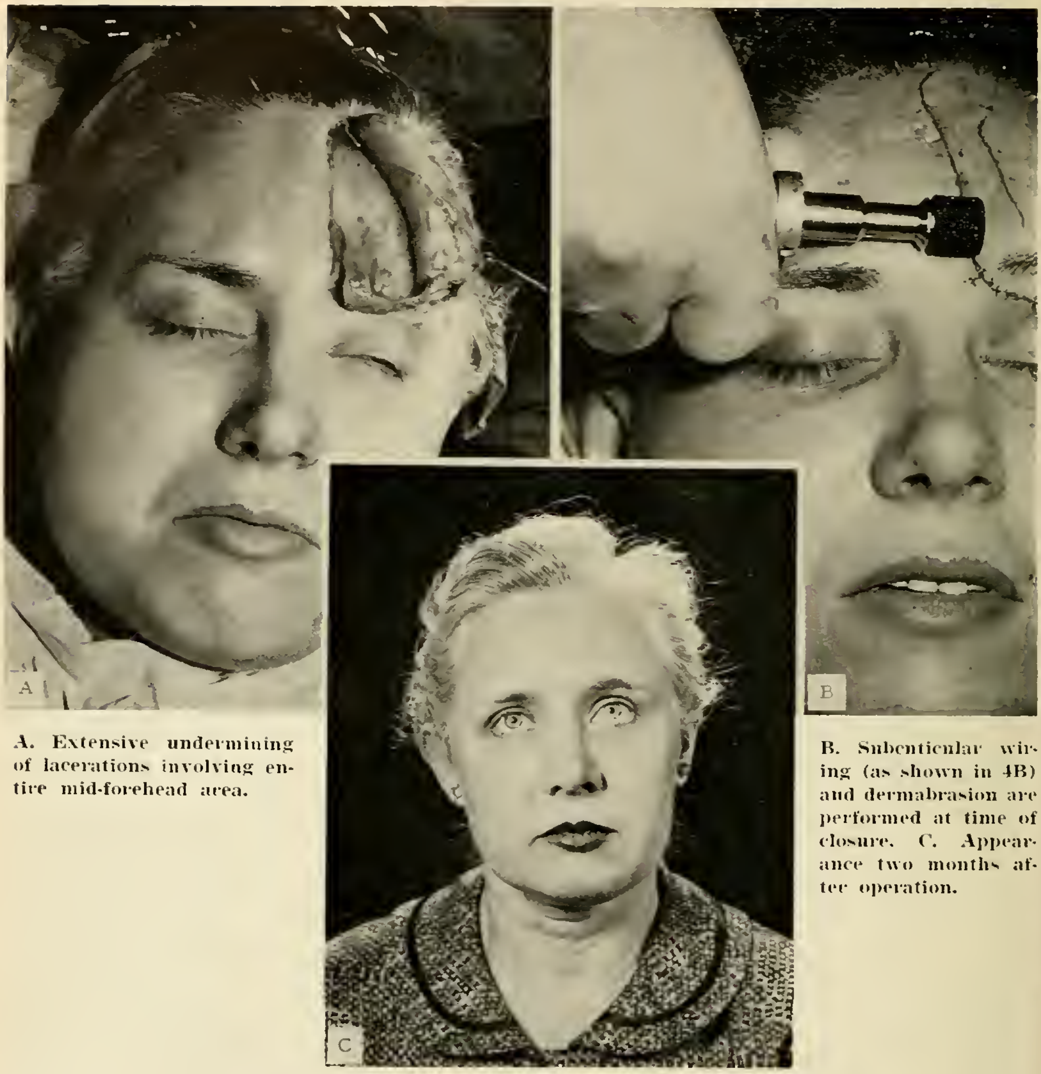


Fig. 4. A. Appearance of avulsing injury two hours after accident. B. A running subcuticular suture of stainless steel (1) is used to minimize tension on skin edges; inverted 4-0 plain catgut sutures (2) are used in the deeper layers. C. A dermabrader is used to level the skin edges. D. View of laceration six days following repair, with wires still in place.



A. Extensive undermining of lacerations involving entire mid-forehead area.

B. Subcuticular wiring (as shown in 4B) and dermabrasion are performed at time of closure. **C.** Appearance two months after operation.

Figure 5

ated for viability before excising. Many questionable areas can be saved if the flaps are carefully debrided and returned to their original position. Exposed bone should be covered by adjacent soft tissue. Knowledge of the normal crease lines of the face is most useful in debriding and closing multiple lacerations (fig. 1).

7. *Closure.* The lacerated tissues are closed in layers and held in position with 4-0 plain catgut. The 5-0 plain catgut sutures may be used for the more superficial layers, the knot being inverted into the wound. A small rubber band or dam drain is usually inserted at the time of closure and gradually withdrawn over a period of two to three days. An untied suture placed in this site is used to approximate these tissues when the drain is removed, in order to minimize scarring.

The wound edges are approximated by one of two methods. Interrupted 6-0 nylon

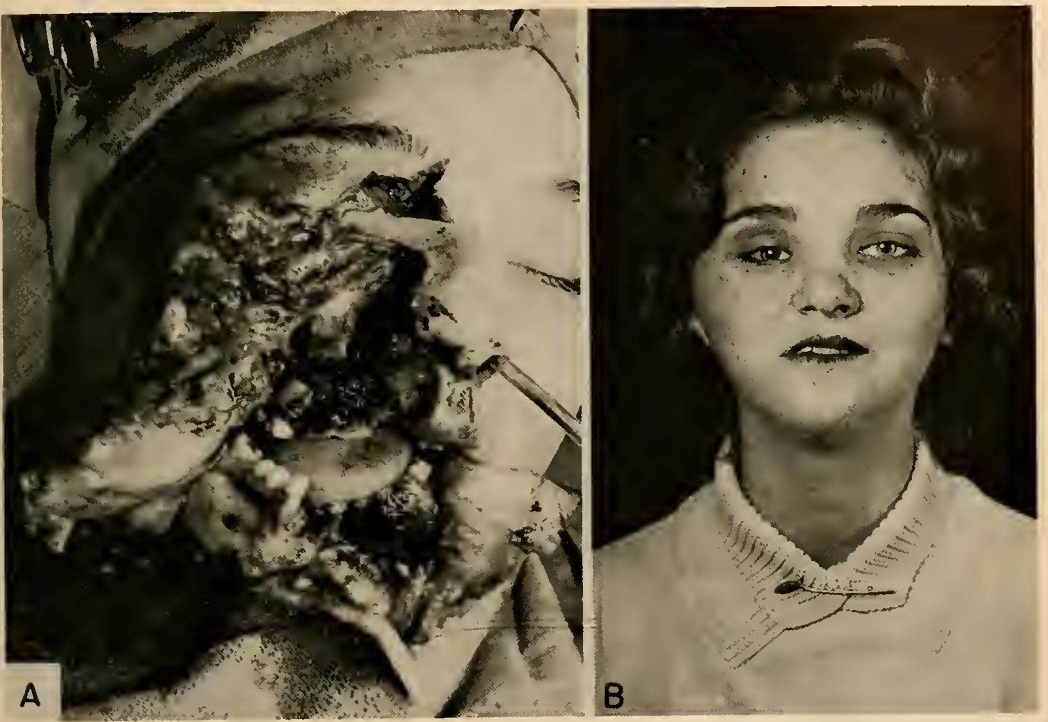


Fig. 6. A. Compound comminuted fractures of the facial bones with extensive avulsing soft tissue damage. B. Photograph taken six months after initial reconstruction. Further revision will be undertaken as maturity of soft tissues warrants.

sutures or the newer polyethylene or Dacron sutures can be used, beginning on the fourth postoperative day (fig. 2); or a 35 gauge stainless steel wire can be used intradermally (subcuticular). This eliminates any small suture lines on the skin and can be kept in position for seven to ten days before removal. I prefer this procedure whenever possible. In wounds over 1½ inches in length, the wire suture should be terminated through the skin and started again in order to facilitate its removal. Any slight irregularities of the skin edges can be dermabraded, or small fine sutures of 6-0 nylon can be used for approximation. These sutures are removed on the second or third day (figs. 4 and 5).

The more complicated wounds, with associated compound comminuted fractures of the facial bones, are managed in the same manner, except that the underlying facial bone fragments must be approximated to their normal anatomic relationship and immobilized before closure of the overlying soft tissue defects (fig. 6).

8. *Dressings.* The management of the traumatized area following satisfactory closure contributes considerably to an eventually successful result. The first postoperative dressings, if the areas are extensive and drains are inserted, should consist of a bulky dressing with occlusive qualities supporting the tissues in the desired position. This will maintain the tissues in close approximation and also serve to minimize the tension on the suture line. One of the newer microbial agents, Triburon, is applied on gauze along the suture line and drain sites. This colorless material minimizes the possibility of contamination and also allows the dressing to be changed without the necessity of detaching the sutures.

After removal of the drains a lighter but still quite effective supportive dressing can

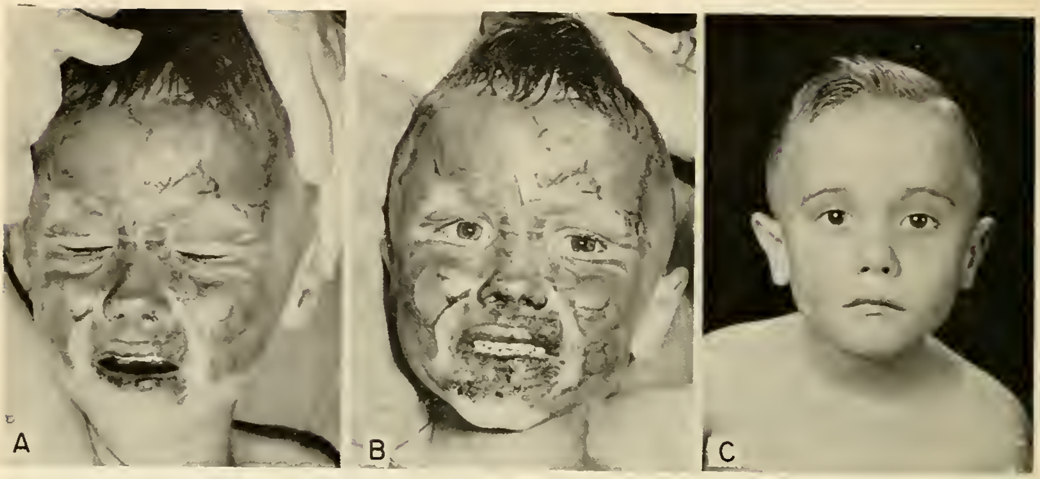


Fig. 7. A. Photograph of face immediately after deep second degree burn. B. Appearance of face following spraying of povidone-iodine solution for 10 days. C. Photograph showing complete healing of burn.

be substituted. This consists of collodion applied to a portion of a roll of gauze, previously cut to fit over the area, after a narrow strip of impregnated gauze has been placed over this suture line and covered by a strip of dry, cotton-filled gauze. This dressing should be maintained over the repaired area, even though the sutures have been removed, for an additional two weeks in order to minimize the postoperative tension on the scarred area. (The patient can also be supplied with scotch tape to cover the area.)

9. *Dermabrasion.* Never techniques involving the use of abrasive wires and wheels have been used to advantage in equalizing any small irreqularities on either side of the incised wound at the time of closure. A light abrasive will often help to minimize scarring, and can also be used to abrade older scars that are being revised. The use of abrasive wheels is particularly successful when the subcuticular wire is used, and does not interfere with removal of the wire on the seventh or tenth day (figs. 4 and 5).

10. *Facial burns.* The burned areas should be cleared early of all loose and devitalized tissue. Afterward the burn is treated by means of the exposure method. We prefer to spray the area with a potent bacterio static agent, Betadine* spray, a povicone-iodine water-soluble solution which allows a sterile film to form over the burned areas. Second degree burns will heal without difficulty under this treatment, minimizing the possibility of third degree loss because of infection. The spray is usually applied two or three times a day to the entire face, the eyes being protected during the procedure. In about 10 days the crust begins to peel off, revealing a pink, nicely healed underlying surface (fig.7).

Summary

The fundamental principles of managing facial wounds should be followed closely. The more meticulous the technique—including the use of fine instruments and suture material, with closure of wounds without tension—the more gratifying the eventual result.

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*Povidone-iodine, Tailby-Nason Company, Delaware.

Health Unlimited

Where It Begins . . . Where It Ends . . . Whose Problem and Responsibility To Keep It Going

ELIAS S. FAISON, M.D.
CHARLOTTE

The subject of this paper is a broad one, and entails a great deal of thought and planning and unlimited cooperation between all medical groups, not only in this nation but throughout the world. Since we believe that this nation leads the world in medical care, however, I will confine my remarks to these shores.

Where It Begins

It would appear that health begins with birth, yet it actually precedes birth. Various research teams have learned that a well balanced diet and healthy surroundings are essential for a healthy population. In the Indian era it was impractical, and quite impossible, for a tribe to halt and wait for a squaw to recuperate from the birth of her child; so the squaw would have to have her papoose and catch up with the march. Medical historians apparently forgot this fact. A number of us can well remember when mothers were kept in bed for a week or more *post partum*. Now ambulation is ordered almost at once. What a mistake was made! One wonders at the morbidity that might have been prevented. It is useless, however, to dwell on this subject, so let us pass on to the art of delivery.

What has happened to this facet of health unlimited? Volumes have, and will be, written about it, but in simple terms, health teams of nurses, doctors, and even midwives have greatly reduced natal mortality.

Now suppose we have a healthy mother and child: What happens then? The pediatrician moves in and prescribes the correct diet, gives vaccines, and so forth, and we are off to a good start. It was in the year 1912 that this state had its first full-time pediatrician. This, then, is the beginning of a life that now has an expectancy of more than 69 years, as against 49 years at the turn of the century.

These to me are truly amazing figures, and one is moved to reflect as to how and why so great a change came about. Our forefathers are to be commended for doing a good job, but they did not intend for us to become complacent. Today we owe it to our progeny, as they did to theirs, to continue to expand life expectancy. How is this to be done, and how is it to continue? In my opinion, there is only one answer: strict cooperation between all member groups of the health team; some government aid—yes, but by all means not government control.

The health team in this country now includes about two million persons. This means that I out of every 88 people devotes his entire time to the health of this nation. I do not have figures for other countries, but I would venture a guess that ours lead them all; and I would remind you that this progress has taken place under a system of free enterprise.

The Nursing Profession

As we consider the membership of the different groups that make up the health team, let us begin with nursing, for we as doctors realize that the health of this nation could not prosper were it not for the nurses. In this connection I would like to quote from the talk by Dr. J. J. L. Blasingame at our meeting in Chicago in February.

"The relationships between physicians and nurses locally, particularly in the hospital and at the community level, are generally good. At the state level, medical-nursing profession relationships are better in some areas than in others. At the national level, the relationship between the two health professions has not been satisfactory for a variety of reasons.

"Periodically, resolutions have appeared in our House of Delegates indicating a variety of problems in the nursing services to patients. These resolutions more often than

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Read before the North Carolina Committee on Patient Care, April 21, 1961.

not have arisen as a result of local tensions. The authors often appear to lack an overall recognition of the problems involved and ask the A.M.A. to do this or that to try to solve the alleged problem.

"Dr. Nafe reported on the history and activities of this Committee for Liaison with National Nursing Organization for the Board of Trustees to the House of Delegates in Miami, and in brief the House approved three recommendations: (1) Use of A.M.A. publications to inform the profession of the facts of the nursing situation; (2) Continued activity in nursing education and recruitment programs; (3) Continued liaison at both policy and staff levels with national nursing organizations. In brief, the Reference Committee on Miscellaneous Business reported that: (1) Physicians should fulfill their obligation of furnishing adequate nursing services by serving on nursing advisory committees; (2) Cooperation of each group in the interest of good patient care; (3) Continuance of liaison at all levels—state, county, and medical staffs of hospitals.

"It is my personal conviction that the medical profession should accept the position that nurses have attained a professional status—in a sense separate and distinct—and in our work we as physicians and the A.M.A. as a medical organization should carefully assume the attitude of co-workers and fellow team mates. We should carefully avoid the appearance or posture of the master-servant relationship; it does not exist and cannot exist.

"Further, it is my considered opinion that we must endorse the nurses' aspiration to raise their standards. Such an attitude on our part will create a more cordial atmosphere, and is certainly in keeping with our standards towards our own profession. There is room for improvement in nursing education, compensation, and service. If the standards are raised, a higher quality of applicant will seek this profession, which is becoming more and more pivotal and responsible as a member of the health team.

"Finally, I believe we need to recognize that the nursing profession has become somewhat stratified—from maid to aide on up to the highly trained nursing educator. Our program of liaison, recruitment and study must deal, it seems to me, with all such various levels of nursing care. This liaison with the nurses can be done largely at the staff level. I am of the opinion that the American Nurses' Association should be looked to, and held responsible, as the overall most representative national group, although we must recognize that it will be necessary at this time to have direct liaison with other national nursing organizations as well. In my judgment, this Committee should not concern itself with day to day routine details of this liaison, but should devote its time and attention to policy problems which staff uncovers, and the Association will need advice and guidance. These problems may not only arise as a result of staff activity, but may come from resolutions to the House of Delegates, from Councils and Committees of the Association, or from inquiries on expressed aspirations or policy problems from national nursing organizations."

Number and distribution of nurses

In 1958 there were 4,600,000 nurses employed in the United States. More than half of the active professional nurses are engaged in hospital nursing. Private-duty nursing is the second largest field; office nursing, public health, occupational health, and nursing education follow in that order. Ninety-nine out of every hundred nurses are women; 55 per cent are married; 65 per cent are less than 45 years of age.

In 1958, of 25,959 nurses surveyed, 0.9 per cent held master's degrees and 5.5 per cent held bachelor's degrees. In 1957, in North Carolina, 543 nurses or 1 out of every 8 were employed in public health work, and 200 were employed full time in industry.

In 1959 there were 8,412 professional nurses serving in our armed forces.

The parent organization of the professional nurse is the American Nurses Association. In 1959 this organization had a dues-paying membership of 173,242. In North Carolina there were 3,667 members.

The National League for Nursing, which works closely with the A.N.A., had, in 1958, 22,550 members. In North Carolina there were 418.

The National Student Nurses Association is an organization of undergraduate nurses that in 1959 numbered 81,029 members. In the same year there 2,225 members of this organization in North Carolina.

In 1959 there were in the United States 79,545 licensed practical nurses. Their organization is called the National Federation of Licensed Practical Nurses, and in 1959 it had a membership of 27,016. In 1958-1959 there were 23,116 admissions to practical nursing programs.

In 1958 there were 27,594 orderlies in this nation.

The International Council of Nurses, with members in 46 countries throughout the world, is a nonpolitical, self-supporting organization dedicated to the development of international understanding between nurses. The purpose of this organization is to improve standards of nursing, nursing education, nursing service, and the socioeconomic status of nurses. It has what is known as an exchange of privilege program whereby it assists nurses in securing temporary employment. It arranges observation trips in other countries and confers many other less important favors. In 1959 there were 190,000 members of this council in the United States. In Great Britain there were 62,000, in Canada 168,000, and in Japan 31,000, etc.

This organization is to the nursing profession what the World Medical Association is to the medical profession. If you will pardon a personal reference, my wife and I greatly enjoyed attending the meeting in Istanbul about three years ago and the one in Copenhagen the following year. It was most pleasant to meet with members from so many nations, all with one motive in mind: to elevate and standardize medical service throughout the world, in a common effort to improve patient care, and at the same time to oppose as an organization interference by the state with regard to the patient-nurse-doctor relationship.

Nursing schools

Now a word about our schools: In 1958 there were 1,126 state-approved nursing schools in the United States, with an average of 181 students each. Six hundred ninety-five had private support, 268 had public support, and 199 received support from both sources. In 1958 North Carolina had 36 approved schools of nursing.

In 1959 there were 607 practical nursing programs approved by the officially designated licensing authority. In North Carolina, in 1958, there were 12 such programs approved.

In 1957-1958 there were 17.2 graduates of basic professional programs and 7.1 graduates in practical nursing for every 100,000 population.

Other Medical Statistics

In 1958 there were 226,830 physicians in active practice and an additional 14,000 osteopaths in the United States. With a population of 176,912,000, this makes 136 physicians for every 100,000 people.

In 1958 there were 91,000 dentists, or 52 for every 100,000 population.

In North Carolina, in May, 1959, there were 3,991 doctors. In the same year there were 1,316 dentists.

Now a closing word about hospitals:

In 1958 there were 6,786 hospitals in the nation. In the same year there were 23,697,117 admissions. This would roughly average 1 to every 7 persons—multiple admissions, however, would alter this figure.

In this state, in the same year, there were 174 hospitals with 631,493 admissions from a population of about 4.5 million. This means that 1 out of every 7 people entered the hospitals in this state, leading to the deduction that North Carolinians are no healthier than the people of the nation as a whole.

It is interesting to note that in 1940, 27 per cent of the students who entered nursing schools withdrew before graduation. In 1958 this figure had risen to 33. I do not have statistics for all the colleges, but I am told by the registrar at Davidson College that about 25 per cent of the students withdraw before graduation.

Where It Ends

In the beginning of this paper the birth of a nation was mentioned. Now where does it end? Life now ends on an average at about 70 years, but where will it end at the turn of the century? I say in all frankness that, in my opinion, this figure will continue to rise until the age of 100 or more will be commonplace. If this happens it will, of course, pose a problem. There are today about 25,000 nursing homes in the nation, and this number is inadequate. All over the nation today voluntary committees on aging are being formed. Civic organizations, religious groups, and the like are sponsoring committees to study the problems that face the aging. The A.N.A. has issued a statement of standards for nursing care in nursing homes.

When one who has been in the health field for the past 25 to 30 years reflects a bit, he is, by compulsion, amazed at the progress that has been made in the care of patients. Laboratory methods and techniques have made great strides. Radiation has made great progress. Tranquilizers and electroshock have made good citizens out of mental derelicts. Cardiovascular surgery has rehabilitated thousands of people who would otherwise be a burden to their families or subjects of the state. Antibiotics have almost conquered infectious diseases. Tuberculosis and polio are on the run, and the common cold is being attacked on every side.

Whose Problem and Responsibility

Now in the end, whose problem and whose responsibility is it to keep health unlimited? The 2,000,000 people who devote their entire time to this end are, for the most part, dedicated persons. They are in groups, to be sure—with their own societies, their national and international organizations—from maids to aide, from bookkeeper to teacher, from intern to extern, from technician to physician, from physical therapy to medical therapy, from the nursing home to intensive care—on up and down the line.

These many groups can be compared to a great machine with many cogs, all of which have to perform in a smooth and cooperative fashion if the care of the patient is to be preserved and improved.

Report from The Duke University Poison Control Center

Jay M. Arena, M.D.

IRON COMPOUNDS

The incidence of poisoning by iron salts seems to be related to the status of iron therapy in any given period. Cases of accidental and homicidal poisoning from iron salts were widely reported in the literature of the middle nineteenth century. Reports of accidental iron intoxication did not appear again in print until 1934, and since 1947 they have appeared with sufficient frequency to warrent editorial attention.

Until the last decade of the nineteenth century, iron therapy was widely employed in the treatment of hypochromic anemias due to body iron deficiencies. At the turn of the century a definite change in attitudes toward the value of iron therapy occurred as the result of the belief that inorganic iron was not absorbed from the gastrointestinal tract. The small doses of iron salts then used had little therapeutic effect, and the value of iron fell into disrepute until the second decade of this century. Then the two fundamental principles of iron therapy previously set forth by Sydenham and Blaud—namely, the value of large doses and the greater potency of ferrous salts—began to be reconfirmed through use.

In recent years ferrous sulfate has been used extensively for the treatment of iron deficiency anemia, and some blood banks give the tablets routinely to donors. The more widespread use of this compound has increased the opportunity for accidental poisoning in young children. Coloring and candy-coating this medication have made it appeal to small children, thereby increasing the hazard. The magnitude of the danger is evident in the vital statistics on fatal accidental iron poisoning which have been recorded in the United States and Great Britain in recent years.

The mechanism of iron poisoning is not known. Pathologic findings have failed to give a satisfactory explanation for death.

Opinions are divided as to whether the fatal outcome is due to shock secondary to local tissue damage to the gastrointestinal tract, to systemic effects following the passage of large quantities of iron into the blood stream, or to the metabolic effects of absorbed iron resulting in respiratory collapse.

One of the unique features of iron metabolism is that iron has no organ of excretion. Iron released from the catabolism of iron compounds is not excreted and must be fed back into anabolic iron pathways. Overloading with parenterally administered iron is not compensated for by increased fecal or urinary excretion. Iron found in the feces represents only unabsorbed iron, and the intestine has no power to regulate, through excretion, the amount of iron in the body. Urinary excretion of iron is negligible, and increased ingestion of iron is not accompanied by elevated urinary ecretion. Thus the capacity to regulate the amount of iron in the body lies in the absorption mechanism.

Iron is absorbed by the mucosal cells of the intestinal tract, and more readily in the ferrous than in the ferric form. How iron passes through the mucosal cell and into the blood stream is unknown. The absorption of iron is not a question of simple diffusion across a membrane, but involves a metabolic transport through the intestinal mucosa. Presumably, the mucosal cell regulates the rate of absorption from the gastrointestinal tract, and seems to act as a barrier to the rapid entrance of iron into the circulation.

The pathologic picture is usually that of hemorrhage and necrosis of the stomach and intestinal mucosa. The liver is frequently involved, showing mild cloudy swelling to small areas of complete necrosis. Among some survivors, fibrosis of the liver and pyloric stenosis have been described. In most cases there has been a marked dilatation of the right side of the heart, with pulmonary congestion and occasional hemorrhage. There have not been any particularly abnormal findings in other organs of the body.

There are three critical phases in iron poisoning: an acute phase of shock that occurs within a few hours after ingestion; a recurrent phase that appears 20 to 48 hours after ingestion; and occasionally a late phase of gastric scarring and contracture, with pyloric obstruction appearing about a month later as a result of the corrosive action of iron salts.

Death has occurred from the ingestion of ferrous sulfate in doses ranging from 40 to 1,600 mg. per kilogram of body weight. The fatal dose for a child 2 years old or younger is estimated to be 900 mg. per kilogram of body weight. Determination of the serum is the test for confirmation of diagnosis and for detecting severity of acute poisoning.

The principal manifestations of poisoning are vomiting and circulatory collapse. Lethargy, vomiting, a fast, weak pulse, low blood pressure, and coma appear within one-half to one hour after ingestion. These symptoms may disappear after four to six hours, followed by a 6 to 24 hour asymptomatic period in which the child seems to improve rapidly. A second crisis occurs with cyanosis, vasomotor collapse, pulmonary edema, coma, and death within 12 to 48 hours.

Treatment

The treatment is directed toward removing the iron and combating shock, and has to be immediate, vigorous and sustained. Teamwork is necessary to carry out the following procedures:

1. Clearance with suction and maintenance of an open airway.

2. Control of shock with available intravenous fluids, blood, plasma, and oxygen.

3. Gastric lavage with a concentrated solution of sodium bicarbonate, 5 per cent disodium phosphate duohydrate, or milk, until the returning fluid is clear.

4. Retention in the stomach of 100 to 300 ml. of one of the above solutions. A demulcent such as egg white may be added to the bicarbonate or phosphate solution.

5. Rectal lavage with one of the above solutions, if there is diarrhea, hyperperistalsis of the lower intestinal tract, or a lapse of three hours or more since ingestion of the iron.

6. In critical cases, oral administration of half the total intravenous dose of edathamil, 35 to 45 mg./kg./24 hours, may be indicated. The effectiveness of oral edathamil in acute iron poisoning, however, remains to be confirmed.

7. Collection of blood for a complete cell count, typing, and crossmatching; for blood pH, iron, sodium, potassium, chloride, and carbon dioxide determinations, as well as for liver function tests.

8. Critically ill patients should receive edathamil intravenously. If no edathamil has been given orally, the intravenous dose should be 70 to 80 mg./kg./24 hours in dextrose or normal saline solution in an 0.5 to 2 per cent concentration. If oral edathamil is used (and the rate of its absorption through a gut wall damaged by iron is not known), only half the above dose should be given intravenously—35 to 40 mg./kg./24 hours.

9. After these emergency measures have been carried out, roentgenograms of the abdomen should be taken for visualization of radiopaque iron tablets in the gastrointestinal tract.

10. Collection of all urine for early detection of possible oliguria and study of the urine sediment for evidence of renal tubular damage.

11. Administration of prophylactic antibiotics, particularly if aspiration of foreign matter into the airway has occurred, and of multiple vitamins which should include vitamin B complex.

12. Guided by the clinical picture and the daily iron levels in serum, and if measurements are available, by urinary output of edathamil and iron, intravenous and/or oral edathamil is continued in a total daily dose of no more than 70-80 mg./kg. The duration of treatment with this drug should not be, and need not be, longer than five days.

13. Follow-up liver function tests and study of the gastrointestinal tract with a radiopaque medium for strictures.

Local Druggist Gets Vote of Confidence

When they have to buy prescription drugs for themselves or their families, American homemakers rely almost completely on their local druggist, according to a survey of women homemaker-delegates assembled in Chicago this week (Sept. 24-28) for McCall's Fifth Annual Congress on Better Living. Ninety-five percent of the 65 women representing America's families at the Congress said they always had prescriptions filled at the local drugstore or pharmacy; only two percent utilize the drug departments of departments stores, and not one had ever had a prescription filled by a drug discount house.

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NOVEMBER, 1961

EXECUTIVE COUNCIL MEETING

The fall meeting of the Executive Council on October 1 in the Carolina Hotel at Pinehurst followed the three-day conclave of the Society's six commissions. The 24 items on the agenda furnished a full day's work—especially since item 21 included the six commission reports. Only the high lights of the meeting will be reported here.

Dr. T. S. Raiford, chairman of the Blue Shield deputation appointed to request the Board of Trustees of the National Blue Shield Association to approve the Hospital Care Association as a Blue Shield agent, reported that the deputation appeared before the National Association in Chicago on Saturday, September 16, and presented a strong plea for inclusion of the Hospital Care Association. The committee had not received a formal answer from the National Association. He stated that Mr. Herndon, executive vice president of the Hospital Care Association, had been notified that the request was denied. This action was quite disappointing, since Hospital Care Association has met every requirement of the State Society. Dr. Raiford stated that it was apparent that the National Association favored a merger of the two groups.

Dr. Amos Johnson commented that he does not want the question of a merger to be the graveyard for extending medical care to the people of North Carolina. He then offered the following resolution: "That the deputation committee be reactivated and made an *ad hoc* committee to pursue the matter of Blue Shield coverage to an ultimate conclusion." This motion was carried unanimously.

Dr. Wayne Benton reported for the Finance Committee that the Society has a surplus of $3,000, and that the investments of the Society's funds had yielded 14 per cent in dividends.

On Dr. George W. Paschal's motion the Executive Council approved the policy of allowing qualified nurses and hospital personnel to give transfusions and intravenous fluids and medication, when they are authorized by the physician in charge of the case. The motion was passed unanimously. In discussing the matter, Dr. Paschal commented that qualified hospital personnel should be trained to use such emergency measures as blood transfusions and intravenous fluids. His Committee on Emergency Medical and Military Service expects to take up with local medical groups planning in North Carolina's 800 corporate communities for carrying out emergency measures in case of a nuclear attack. His committee expects to get out a booklet on preparing for emergencies.

Dr. John Rhodes reported for his Committee on Arrangements for the Annual Convention, to be held again in Raleigh. He stated that the same general plan that was used at the 1960 meeting would be followed, with only a few changes .One change was that the President's Ball would be held in the Sir Walter Hotel instead of the Student Union.

The Committee on Scientific Awards recommended that the chairman of this committee be appointed by the State Society president, instead of elected by the committee. This would give him a longer time to arrange for the committee's work. The committee also suggested that copies of papers selected for awards be sent to all members of the committee at the same time, instead of being passed from one to another. Another recommendation was that the section scientific programs be included in the official program of the annual convention.

Mrs. George T. Noel, president of the Auxiliary, was in Chicago attending a meeting of the State Society Auxiliary presidents. Her report was read by her husband, and as usual was well received. The Auxiliary continues its constructive program of community service, cooperation with our State Society's Legislative Committee, and promoting training for careers in health.

Dr. Ralph Garrison's discussion of the American Medical Education Foundation was important enough to justify a separate editorial.

The Committee on Public Relations offered a recommendation similar to the one adopted by the Georgia Medical Association. The recommendation was that each county medical society be requested to go on record publicly by a resolution in support of the American Medical Association as representing satisfactorily a majority of its membership. Copies of any resolutions passed by county societies are to be sent to their respective congressmen and senators, and to State Medical Society headquarters office. In addition, members were encouraged to write directly to their congressmen and senator, expressing confidence in the A.M.A.

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DR. H. STUART WILLIS NEW PRESIDENT OF THE N.T.A.

Dr. H. Stuart Willis, superintendent and medical director of the North Carolina Sanatorium System and clinical professor of medicine at the University of North Carolina School of Medicine, was installed as president of the National Tuberculosis Association at its recent annual meeting. Dr. Willis richly deserves this recognition. He is a past president of the American Thoracic Society and of the North Carolina Tuberculosis Association, and for several years has been chairman of the Committee on Medical Research of the N.T.A.

This Journal congratulates Dr. Willis on this signal honor—and also congratulates the N.T.A. in making such a wise choice.

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AMERICAN MEDICAL EDUCATION FOUNDATION

Some years ago the American Medical Association took one of its most constructive actions, when it sponsored the American Medical Education Foundation, for the purpose of raising funds for our medical schools. All money contributed to the Foundation is given to the medical schools of the nation with no strings attached. It affords doctors now in practice the opportunity of repaying society for the part of their medical education that was not covered by tuition fees.

Dr. Ralph Garrison is the state chairman of the AMEF. His report to the Executive Council gave North Carolina doctors cause for both pride and embarrassment: pride, because in 1959 North Carolina was fourteenth in the nation in contributions to medical schools; embarrassment, because it was forty-eighth in contributions to the American Medical Education Foundation. Many doctors do not know that they can contribute to their alma maters through the AMEF by designating their gifts for the school of their choice. In 1959 all four-year medical schools were given $4925 of undesignated funds: all two-year schools. $3462. In 1959, 188 North Carolina doctors gave $6325.84 to the AMEF, while 1589 doctors gave $101,493.73 directly to the three medical schools in the state. Simple subtraction indicates that only $1400 was designated for medical schools through the AMEF, while more than $100,000 was given directly.

The AMEF is the best answer yet to the threat of federal control of medical education. It would present a far more effective argument if all funds were channeled through it, and a loyal alumnus could give just as much to his alma mater. Since the

A.M.A. meets all of the foundation's expenses, every dollar contributed goes directly to medical schools. A contribution to the AMEF can be deducted for income tax purposes, just as direct contributions to medical schools can be. Let us hope that North Carolina will raise its standing in AMEF contributions to the level of its contributions to all three medical schools.

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WINDOW DRESSING

An editorial by Dr. Benson Harer in the *Pennsylvania Medical Journal* for September makes a point that needs to emphasized. It is so pertinent that it is quoted, in part, below.

"Even though their own professional fees have risen only moderately, physicians have been severely criticized and blamed for the increased cost of health care. This is probably due to a grossly exaggerated opinion on the part of many people of the doctor's ability to control health care costs. Actually, the physician has direct control only of his own charges and a degree of indirect control over certain other components of health care such as drugs, admission to and length of stay in hospitals, and diagnostic tests and treatment procedures ordered by him. Nevertheless, the medical profession is being viciously attacked and our image in the public eye is suffering from these attacks.

". . . These problems can be solved only through full realization and acceptance by all doctors of their responsibility to provide no more than needed medical care, high in quality and at affordable prices.

"It is not necessary, in this discussion, to dwell on unnecessary surgery, prolonged stay in hospitals, and other obvious and controllable factors in rising health care costs since, with one exception, all of these have been exhaustively reported many times. The exception, in this writer's opinion, is that of window dressing. By this is meant the practice followed by a small percentage of doctors of ordering drugs, laboratory tests, and treatment procedures that add little or nothing to the welfare and comfort of the patient but are ordered solely to impress the patient with the physician's scientific attainments and concern for him.

". . . We are referring . . . to the routine use of drugs, laboratory tests, and treatment procedures that are not indicated but are used by the physician for ulterior purposes such as to justify a higher fee, to build practice, or even to conceal a lack of true professional competency . . .

"Factors completely beyond our control are causing a steady rise in the cost of health care. It is imperative, therefore, that all controllable factors of cost be actually controlled if we are to avoid socialized medicine with its accompanying federal control of not only costs but actions of doctors. Window dressing is a controllable factor and must be eliminated. Overtreatment of patients cannot be tolerated any more than undertreatment. Sooner or later, the window dresser is exposed and shown for what he is. Modern scientific medicine has so much to offer that competent, conscientious doctors do not need to resort to the phony tactics of window dressing."

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BEAUTY THROUGH BIOCHEMISTRY

The same issue of the *New York Times* that told of the tranquilizing power of pearls dangled an even more alluring bait to the female of the species. A full-page advertisement of Mr. Gayelord Hauser's latest book made the provoking claim that it would enable the reader "to develop a bloom of prize-winning beauty" by learning "how to control the chemical balance of your body." The secret of being beautiful, Mr. Hauser implies, is in having one's chemicals properly balanced: "Remember: the most beautiful woman in the world is made of the very same chemicals as you. The difference is in the way those chemicals are distributed throughout the body."

The advertisement does not overlook the possibility of gaining the attention of the male. A "special" for the husband advises: "Teach him to exercise his scalp and save his hair. Reveal to him the connection between diet and sexual potency."

It is quite probable that this book will join "Arthritis and Common Sense" and "Folklore Medicine" in the best-seller list.

Bulletin Board

COMING MEETINGS

North Carolina Academy of General Practice, Annual Meeting — Carolina Hotel, Pinehurst, November 26-29.

University of North Carolina Postgraduate Medical Course — Memorial Mission Hospital, Asheville, each Tuesday for six weeks; Grace Hospital in Morganton, each Wednesday for six weeks.

Duke Resident Lectures in Ophthalmology — Eye Clinic, Duke Hospital, Tuesday evening at 7:30 p. m.

North Carolina Mental Health Association Meeting — Jack Tar Hotel, Durham, February 16-17, 1962.

Nineteenth Annual Watts Hospital Medical and Surgical Symposium — Watts Hospital and Jack Tar Hotel, Durham, February 23-24, 1962.

American Medical Association, Clinical Meeting — Denver, Colorado, November 27-30.

Southern Surgical Association, Annual Meeting — The Homestead Hotel, Hot Springs, Virginia, December 5-7.

NEW MEMBERS OF THE STATE SOCIETY

The following physicians joined the Medical Society of the State of North Carolina during the month of September, 1961:

Dr. Hoke Smith Nash, Jr., 106 West 7th Street, Charlotte; Dr. John Edwin Drew, Box 176, Macclesfield; Dr. H. Mac Vandiviere, N. C. Sanatorium System, Chapel Hill; Dr. Ralph Guy Brashear, S. Main Street, Wendell; Dr. Clabe Webester Lynn, Jr., 2006 Fairview Road, Raleigh;

Dr. Andrew Harper McElroy, Jr., River Street, Box 98, Colerain; Dr. William Robert Purcell, 418 King Street, Laurinburg; Dr. Campbell White McMillan, 418 King St., Laurinburg; Dr. Paul Maynard Lerner, 309 Doctors Bldg., Asheville; Dr. Theodore Dennis Scurletis, 811C Daniels St., Raleigh; Dr. Joe Alexander Bain, 604 Banks Avenue, Goldsboro.

NEWS NOTES FROM THE UNIVERSITY OF NORTH CAROLINA SCHOOL OF MEDICINE

On October 24 the U.N.C. School of Medicine inaugurated a new kind of medical postgraduate program, supported in part by a grant-in-aid from Merck Sharp and Dohme. The program involves the use of FM radio for broadcasting educational programs to physicians meeting as organized groups in local hospitals, with provision being made for these groups to ask questions of the speakers through a conference telephone hookup.

Twenty-four weekly programs are planned for this year, to be broadcast on Tuesdays from 1:00 to 2:00 P.M. They consist of a 30-minute presentation and 30 minutes of questions and discussion. While only those in the organized groups can submit questions, any physician who lives within the effective range of WUNC-FM (about 100 miles) and who has an FM receiver can tune in and listen. WUNC-FM will be found at 91.5 megacycles on the FM dial.

There will be sequences of three or four programs on related subjects of interest to all physicians. Participants will include guests from within and without the state as well as members of the U.N.C. faculty. Once under way, detailed programs will be published in What Goes On and the North Carolina Medical Journal.

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The National Foundation—March of Dimes has awarded a grant of $30,456 to the U.N.C. School of Medicine to continue support of the school's rehabilitation teaching program.

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Research on bleeding diseases underway in the Department of Pathology has been cited by the Southern Regional Education Board in its publication "University Research—Medical Milestone." The publication includes the department's research on hemophilia and leukemia along with other items gathered from medical schools in the SREB's 16-state region.

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The U.N.C. School of Medicine has been awarded a grant of $39,773 from The National Foundation—March of Dimes for renewed support of the University's program for teaching physical therapists.

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Harry Gooder, Ph.D., and Wallace A. Clyde, M.D., have been appointed to the faculty of the U.N.C. School of Medicine, it was announced recently.

Born in Castleford, Yorks, England, Professor Gooder attended Leeds University where he received the B. Sc. and the Ph.D. degrees. He has occupied positions of research fellow, Harvard Medical School; research assistant, Central Public Health Laboratory, London; and visting teacher, Chelsea College of Science and Technology, London.

A native of Birmingham, Alabama, Dr. Clyde attended Vanderbilt University where he received the B.A. degree in 1951 and the M.D. degree in 1954. From 1957 to 1959 he was a pediatrician with the United States Naval Hospital, Memphis, Tennessee. He was a postdoctoral research fellow at Western Reserve University from 1959 to 1961.

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The fifth annual University of North Carolina School of Medicine Symposium was held November 9-10, with physicians from throughout the state attending along with doctors from Virginia and South Carolina.

The visiting speaker was Dr. Lewis W. Wannamaker of the University of Minnesota School of Medicine. Other speakers, all of the U.N.C. School of Medicine except one, were Drs. John H. Arnold, Thomas B. Barnett, Wallace Clyde, William

J. Cromartie, Floyd W. Denny Jr., Janet J. Fischer, Sylvanus W. Nye, Robert J. Senior, and John K. Spitznagel.

Dr. Jacob Koomen is with the State Board of Health in Raleigh and also is a visiting associate professor in the U.N.C. School of Public Health.

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The third annual "Medical Science Lecture Series" began at the University of North Carolina School of Medicine on Saturday, September 30, with an international panel of speakers.

This year the series is being directed by Dr. T. M. Csaky, associate professor of pharmacology. The lectures will be given each Saturday morning at 11 o'clock in the Clinic Auditorium of the School of Medicine through December 16, with the exception of the period of Thanksgiving holidays. All interested persons are invited to attend.

The title of the series is "Biological Transport Mechanism—Role of Cell Membrane in Active Transport and Nerve Connection."

A full program of speakers, dates and subjects will be published in the near future. In the meantime, persons who would like to receive this information may write Medical Science Lecture Series, U.N.C. School of Medicine, Chapel Hill.

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Dr. George B. Penick has been appointed an associate professor of pathology, School of Medicine, and Director of the School's research program-project on thrombosis and hemorrhage. He will join the faculty on a full-time basis in February, 1962, at the time the new research program is instituted.

The program is designed to support comprehensive research into the basic nature of disorders of blood coagulation, such as occur in bleeding disorders, as in hemophilia and leukemia, and in thrombosis, such as in coronary heart disease.

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Dr. George Johnson Jr., a native of Wilmington, has joined the faculty of the University of North Carolina School of Medicine as an assistant professor in the Department of Surgery.

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Professors Charles H. Burnett, A. T. Miller Jr., Carl W. Gottschalk, and Associate Professor John H. Schwab, of the School of Medicine, have returned from one-year leaves. Professor Burnett, who is chairman of the Department of Medicine, had a Commonwealth Fund Fellowship for study at the University of London. Professor Miller (physiology) served as Liaison Officer in the Naval Research Branch Office of the Office of Naval Research in London.

Professor Gottschalk (medicine) spent a year studying at the Biochemical Institute of the University of Copenhagen, Copenhagen, Denmark, and Professor Schwab (bacteriology) studied at the Lister Institute of Preventive Medicine, London.

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Dr. Clayton E. Wheeler, Jr., has been named chief of the Division of Dermatology and professor of dermatologic medicine at the University of North Carolina School of Medicine.

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E. B. Crawford Jr., of N. C. Memorial Hospital was inducted as a fellow in the American College of Hospital Administrators Sunday, September 24, at Atlantic City, New Jersey.

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The U.N.C. School of Medicine is holding its first postgraduate medical courses of this academic year in Asheville and Morganton.

The Asheville course began Tuesday, October 3, and will be held each Tuesday for six weeks. The course in Morganton began Wednesday, October 4, and will continue each Wednesday for six weeks.

The co-sponsor of the Asheville course is the Buncombe County Medical Society. The Burke County Medical Society is co-sponsoring the Morganton course.

At both cities two lectures are being given each day the courses are held. In Asheville the lcetures are at 5 and 7:15 p.m. Both of these lectures are held at the Buncombe County Medical Society Library at Memorial Mission Hospital.

The lectures in Morganton are at 4:30 and 7:30 p.m. The first lecture each day is at the Nurses' Home of Grace Hospital. The evening lecture is at the Mimosa Golf Club.

Some of the lectures will be given by faculty members of the U.N.C. Medical School. There will be, however, a number of guest lectures. These will represent the Western Reserve School of Medicine, the Medical College of South Carolina, the Bowman Gray School of Medicine, and Jefferson Medical College.

NEWS NOTES FROM THE BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

Dr. Howard H. Bradshaw, professor and chairman of the Department of Surgery, has been awarded the American College of Surgeons' Distinguished Service Award.

Dr. Bradshaw received his award Thursday, October 5, at the annual meeting of College Fellows at Chicago from Dr. I. S. Ravdin, the College's retiring president.

He received the award for "leadership and support in broadening the scope of the College's activities" and "for his long and distinguished career as a professor of surgery and as an educator for young surgeons; for his devotion to the American College of Surgeons for more than 20 years; and for his esteemed service as a member of the College's Board of Governors as its secretary, and as its chairman."

Dr. Bradshaw has been professor and chairman of the Department of Surgery at the Bowman Gray School of Medicine since 1941.

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Dr. John R. Ausband, associate professor of otolaryngology, was recently elected secretary-treasurer of the North Carolina Eye, Ear, Nose and Throat Society.

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Dr. D. LeRoy Crandell, associate professor of anesthesiology, was recently elected president of the North Carolina Society of Anesthesiologists. Dr. C. Max Drummond, instructor in anesthesiology, was elected secretary-treasurer of the society.

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Dr. Richard C. Proctor, associate professor and chairman of the Department of Psychiatry, was recently chosen president-elect of the Southern Psychiatric Association. He will take office in October, 1962. The association is composed of members from 17 southern states and the District of Columbia. He has served previously as secretary-treasurer of the association.

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Mr. Clyde T. Hardy, Jr., director of the Department of Clinics, was recently chosen president-elect of the National Association of Clinic Managers. He will become president in October, 1962. Mr. Hardy previously has served as second and first vice president, and as chairman of the association's committee on personnel and public relations and the committee on clinic organization.

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Dr. R. Winston Roberts, professor of ophthalmology, spoke to the Los Angeles Ophthalmological and Otolaryngological Society, Thursday, October 5, on the "Diagnosis and Handling of Early Glaucoma." He also taught a seminar course on the same subject at the annual meeting of the American Academy of Ophthalmology and Otolaryngology, October 8-13, at Chicago.

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Dr. Eben Alexander, professor of neurosurgery, participated in a neurosurgical symposium on head injuries at the American College of Surgeons meeting at Chicago.

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Dr. Henry G. Cramblett, associate professor of pediatrics, was a participant in a round-table discussion of pediatric virology during the annual meeting of the American Academy of Pediatrics at Chicago, October 2-6.

News Notes from the Duke University School of Medicine

One thousand persons attended a testimonial dinner for Dr. Wilburt C. Davison, retired dean of the Duke University Medical School, on Thursday night, October 5.

The invitational affair for friends and professional associates of Dr. Davison was held in connection with the University's Symposium on the Commonwealth of Children, October 4-8.

Both the dinner and the symposium honored Dr. Davison, who headed the Duke Medical School for a third of a century and made many important contributions to health affairs at the state, national and international levels.

A series of addresses citing Dr. Davison's contributions in various areas of medical education and health affairs highlighted the dinner program.

Dr. Stanhope Bayne-Jones of Washington, D. C., former Yale Medical School dean and currently a member of the Army Scientific Advisory Committee, was the principal speaker. He discussed the activities of Dr. Davison in the medical profession and, governmental health circles.

George Watts Hill Sr. of Durham, who pioneered with Dr. Davison in providing low-cost hospitalization insurance by organizing the Hospital Care Association (Blue Cross), spoke on Dr. Davison's contributions to community medical care; and Dr. Watson Rankin of Charlotte, former head of the Hospital and Orphans Section of the Duke Endowment, described Dr. Davison's leadership in the Duke Endowment's health activities.

Dr. Jean D. Craven of Lexington spoke on "Dr. Davison as Mentor to the Residents." She was Duke Hospital's first resident in pediatrics under Dr. Davison.

Toastmaster for the dinner was Dr. Paul M. Gross, William Howell Pegram professor of chemistry at Duke and president-elect of the American Association for the Advancement of Science.

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The 1961-1962 series of lectures in ophthalmology began at Duke Hospital on September 9 and will continue through May 5, 1962. All lectures will be given in the Eye Clinic of the hospital.

Resident lectures are being given from 7:30 to 8:30 p.m. on Tuesday evening through May 1. Additional lectures have been scheduled on the following dates:

Saturday, February 10	
Saturday, February 10	7:00-9:00 p.m.
Thursday, May 3	7:00-9:00 p.m.
Friday, May 4	8:00-12:00 a.m.
Saturday, May 5	1:00-3:00 p.m.

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Dr. J. Leonard Goldner, professor of orthopedic surgery at the Duke University Medical Center, has been appointed council-elect of the Southern Medical Association for the State of North Carolina.

Formerly an alternate councilor, Dr. Goldner succeeds Dr. George Wilson of Asheville and assumed his new duties following a meeting of the association in Dallas, Texas, November 6-9.

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Norman K. Nelson, assistant director of the Duke University Bureau of Public Information, has been elected a member of the National Association of Science Writers.

Nelson has served as public information officer for the Duke Medical Center since 1956. He joined the Bureau of Public Information staff in 1949.

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Duke University Medical Center scientist R. Frederick Becker has been given a key responsibility in developing a new medical school in the Far East.

A member of the Duke anatomy faculty, Dr. Becker left for Thailand early in October under assignment from the International Cooperatives Administration of the U. S. State Department. During a two-year leave of absence from Duke, he will head the organization of the Anatomy Department in the Medical School of Chieng Mai. The school is now being established in northwestern Thailand near the Burma border.

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A center for cystic fibrosis treatment, research, and training will be established at Duke University with financial support from the National Cystic Fibrosis Research Foundation, it was announced recently.

Duke has been awarded an initial eight-month grant of $14,000 by the Foundation to support this undertaking, and future support is anticipated.

Dr. Alexander Spock of the Duke pediatrics faculty will head the new center.

Dr. Spock emphasized that the Duke center "is not intended to take the place of continuing care by family physicians. Its primary purpose with relation to patient care is to provide diagnostic facilities not available in every locality."

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A $33,000 grant to study "Continued Care Through the Relation of Hospital Care to Community Resources and Rehabilitation" has been awarded to Duke University by the Office of Vocational Rehabilitation, U. S. Department of Health, Education and Welfare.

Dr. James H. Semans, professor of urology and chairman of the Duke Medical Center's rehabilitation committee, will be the medical director of this program.

Dr. Semans explained that "continued care means the communication between the hospital and local physicians and local agency resources for patient care."

Forsyth County Medical Society

Dr. William H. Zinkham, associate professor of Pediatrics at Johns Hopkins Medical School, was the speaker at monthly meeting of the Forsyth County Medical Society held in Winston-Salem on October 10. His subject was "Drug-Induced Hemolytic Anemias."

Edgecombe-Nash Medical Society

The Edgecombe-Nash Medical Society held its regular monthly meeting in Rocky Mount on Wednesday, October 9. A member of the surgical staff of Duke University Medical Center spoke on a surgical topic.

News Notes

Dr. John H. Cox, has announced the opening of his office for the practice of dermatology and syphilogy in Winston-Salem.

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Dr. Campbell White McMillan has announced the opening of his office at 418 King Street, Laurinburg, for a practice limited to pediatrics.

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Dr. Robert W. Whitener has opened his office at 1305 North Elm Street in Greensboro for the practice of psychiatry.

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Dr. William C. Matthews and Dr. Richard T. James, Jr., of Charlotte have announced the association of Dr. Thomas N. Massey, Jr., in the practice of internal medicine. Offices are located at 217 Travis Avenue, Charlotte.

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The Nalle Clinic, located at 1350 South King Drive, Charlotte, announces the association of Dr. Fitz Gerald Hiestand, Jr., in the partice of internal medicine (gastroenterology).

Augusta Postgraduate Medical Assembly

The Augusta (Georgia) Postgraduate Medical Assembly will be held in that city, under the sponsorship of the Richmond County Medical Society, April 2-4.

The program will be one of general interest and will include speakers in the major fields of medicine.

The registration fee of $15.00 covers the scientific sessions and a cocktail party.

For particulars write Julius T. Johnson, M.D., Chairman, Postgraduate Assembly Committee, P. O. Box 3223, Augusta, Georgia.

American College of Surgeons

About 1,103 surgeons were inducted as Fellows of the American College of Surgeons at the annual five-day Clinical Congress of the organization held in Chicago last month. Fellowship, entitling the recipient to the designation F.A.C.S. following his name, is awarded to doctors who fulfill comprehensive requirements for acceptable medical education and advanced training as specialists in one or more branches of surgery, and who give evidence of good moral character and ethical practice.

Those receiving this distinction from the State of North Carolina at the 1961 convocation are as follows: Lt. Cmdr. Ross M. Lehman, Jr. (MC

USN), Camp Lejeune; A. Stark Wolkoff and Robert Zeppa, Chapel Hill; Lewis E. Curlee, Concord; Marcus L. Dillon, Jr., and Seth G. Hobart, Jr., Durham; Stanford S. Setnor, Fayetteville; Robert B. Groves, Jr., and Edward S. Whitesides, Gastonia;

John E. Lyday, Greensboro; Frank H. Longino, Greenville; James S. Gilliam, Jr., High Point; Howard E. Strawcutter, Lumberton; Stewart M. Scott and William H. Sewell, Oteen; Maurice G. Couturier, Sr., Reidsville; Harry H. Weathers, Roanoke Rapids; John C. Foushee, Sanford; Shelley C. York, Jr., Thomasville; John B. Codington, Wilmington; and Charles C. Parker, Wilson.

American Board of Obstetrics and Gynecology

The Part I Examinations (written) of this board will be held in various cities of the United States, Canada, and military centers outside the Continental United States on Friday, January 5, 1962.

Applicants and candidates for examination in 1963, please note that the deadline date for making application is advanced to July 1, 1962.

Current Bulletins outlining present requirements may be obtained by writing to the Executive Secretary, Robert L. Faulkner, M.D., American Board of Obstetrics and Gynecology Cleveland 6, Ohio.

Association of Military Surgeons of the United States

The Executive Council of the Association of Military Surgeons of the United States has announced the appointment of Major General Thomas J. Hartford, Deputy Surgeon General of the Army, to the position of executive director of the association, effective October 15, 1961.

In announcing the appointment Dr. Leroy E. Burney, president of the Association said: "General Hartford brings to our association his long years of experience in administration and military medical matters. He will give his full attention to the administration of the Association which will permit Colonel Robert E. Bitner to devote more time to expanding the services and influence of the Association as editor of **Military Medicine**, our official journal."

Association of American Medical College

The Association of American Medical Colleges has announced it will begin accepting 1962 applications for the unique foreign fellowship program which offers future American physicians an opportunity for medical study in underdeveloped areas of the world. For the third year, Smith Kline & French Foreign Fellowships are being offered to junior and senior students in U. S. medical schools. Descriptive brochures and application forms have now been mailed to all deans, said Dr. Ward Darley, executive director of the Association. Darley emphasized that the closing date for filing applications is December 31, 1961.

The purpose of the program is to provide selected medical students the opportunity to benefit from unusual clinical experiences abroad. The Fellows are able to study and practice preventive medicine in remote areas of the world where health services are often carried on in primitive surroundings. The medical students help to bring modern American medical procedures to these areas, and at the same time gain firsthand experience with other cultures and peoples.

U. S. Department of Health, Education, and Welfare

The number of poison-control centers affiliated with the National Clearinghouse for Poison Control Centers rose to a new high of 460 as of July 1, according to an announcement by Surgeon General Luther L. Terry of the Public Health Service.

The National Clearinghouse for Poison Control Centers serves local centers by providing information on new products which it obtains through a voluntary arrangement with manufacturers. Over 200 major producers of drugs and household products inform the clearinghouse of the ingredients their products contain and the antidotes for them. The National Clearinghouse is directed by the PHS Division of Accident Prevention, headed by Assistant Surgeon General A. L. Chapman.

[The North Carolina Medical Journal publishes regular reports from the Duke University Poison Control Center in Durham. Dr. Jay M. Arena is director.—Ed.]

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Dr. Luther L. Terry, Surgeon General of the Public Health Service, reported in August that an upswing in the influenza cycle is likely to hit this country during the fall and winter.

He recommended immediate vaccinations for persons in three groups which accounted for most of the 86,000 flu-triggered deaths between September, 1957, and March, 1960. These groups are: Persons with heart disease, pulmonary disease, diabetes and other chronic illnesses; persons over 65; and pregnant women.

"We are probably due for some Asian flu outbreaks, since they come in two to three year cycles, and we are overdue for Type B flu outbreaks which come in four to six year cycles," Dr. Terry said. "Asian flu has been dormant here since March, 1960, and it has been more than six years since we had much Type B flu in this country."

The Public Health Service is alerting physicians and health officers, Dr. Terry said, and the Bureau of Public Assistance, another unit of the Department of Health, Education, and Welfare, is urging state welfare agencies to include flu immunizations in medical care provided under public assistance programs.

Recommendations on use of flu vaccine, including a detailed listing of the chronic conditions most likely to be complicated by influenza, are being furnished to private physicians and health officers. They were prepared by the Surgeon's Communicable Disease Center in Atlanta, Georgia, on July 31. At that meeting, committee members agreed unanimously on the likelihood of outbreaks this winter and on the need for a special effort to immunize high risk groups in advance.

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A new program to provide a broader form of financial support to schools of medicine, dentistry, osteopathy, and public health has been announced by Dr. Luther L. Terry, Surgeon General of the Public Health Service.

"The prime purpose of this program is to increase the capacity of the Nation's research and educational institutions for carrying out their health-related research and research training," Dr. Terry said. "We believe it will meet the needs of these institutions for greater flexibility in the use of portions of the federal support funds they have been receiving."

Copies of the Policy and Information Statement in General Research Support Grants are available from the Information Office, Division of General Medical Sciencies.

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The Food and Drug Administration recently announced a new warning label for phenindione, a synthetic anti-coagulant drug marketed by several drug companies. A number of cases of blood dyscrasias and agranulocytosis have been reported.

There are also reports implicating the drug in cases of hepatitis and hypersensitivity reactions.

* * *

Public Health Service

During the year ending June 30, 1960, illness and injury caused the American people to stay home from work, stay in bed, or otherwise cut down their usual activities for an average of 16 days per person, including six days of bed disability.

These estimates come from the latest in a series of published statistical reports of the U. S. Public Health Service's National Health Survey. They apply to the civilian population of the country exclusive of persons confined to long-term institutions.

VETERANS ADMINISTRATION

Appointment of five new members of the Special Medical Advisory Group of the Veterans Administration has been announced by the VA.

The group, composed of outstanding non-government physicians and specialists in related fields, meets quarterly in Washington, D. C., to advise the Administrator of Veterans Affairs and the VA medical program.

New chairman of the group is Dr. Barnes Woodhall, dean and professor of surgery of Duke University School of Medicine, Durham, North Carolina. Dr. Woodhall had been serving as vice chairman.

* * *

A book that may become a landmark in the study of Parkinson's disease has just been published by the McGill University Press, Montreal, Canada.

Titled "The Shaking Palsy," it is drawn from the symposium on Parkinson's disease held in Montreal in October, 1959, under the auspices of the United States Veterans Administration and the Department of Veterans Affairs of Canada.

Editors of the book are Dr. Blaine Nashold, chief of the neurosurgical service of the VA hospital at Durham, North Carolina, and assistant professor of neurosurgery at Duke University School of Medicine, and Dr. Harold Elliott, neurological suregon-in-charge at Queen Mary Veterans Hospital, Montreal, director of the department of neurology and neurosurgery at Montreal General Hospital, and assistant professor at McGill University.

The two agencies have exchanged medical information for over a decade.

* * *

Dr. Lee D. Cady, director of the Veterans Administration hospital at Houston, Texas, has been awarded the Physicians Award of 1960 by the President's Committee on Employment of the Physically Handicapped.

Noting that scores of handicapped persons are employed at the Houston VA Hospital, Dr. Cady said: "We practice what we preach."

* * *

A new method of treatment for cancer, originated primarily by a Veterans Administration doctor, has produced such good results in early testing that it is being explored against cancers of all types at 24 VA hospitals across the nation.

Known as local infusion, it enables doctors to concentrate large amounts of a cancer-destroying drug amethopterin (Methotrexate), at the site of the tumor, while protecting the rest of the body against the drug by means of an antidote (citrovorum factor).

Doctor Diplomats

Five physicians from Tulsa, Oklahoma, members of the First Presbyterian Church of Tulsa, are giving up their practices for six-week periods to serve voluntarily at the Miraj Medical Center in Miraj, India.

Dr. C. S. Lewis, one of these five Tulsa physicians, recently reported to the A.M.A. on the progress of the project labeled "Doctors in Asia."

The first of the group of volunteer physicians flew to Miraj in mid-August. On his return at the end of September the next doctor will make the trip. In all, the five physicians will donate a total of thirty weeks to the program. The project is endorsed by the Tulsa County Medical Society. Funds for medical equipment, transportation, and other expenses were raised through church and public contributions.

Other groups of American physicians are also becoming interested in the possibility of initiating a similar venture in their own communities. For example, several doctors met with Dr. Lewis during his A.M.A. visit to discuss the feasibility of adopting an overseas program which would provide medical care to another area of the world equally in need of such assistance.

Still another example of American physicians demonstrating their interest and willingness to serve in foreign mission fields on a temporary basis is shown by the large number of doctors who have written to the A.M.A. Department of International Health in the last few months to inquire about such service. This new Department administers a program approved last June by the A.M.A. House of Delegates whereby members of the A.M.A. may volunteer for service in the foreign mission fields on a temporary basis when emergencies arise. Cooperating with A.M.A. in this program are missionary agencies representing every denomination sponsoring American medical missionaries.

Physicians interested in volunteering for such service are asked to write directly to the A.M.A. Department of International Health, 535 N. Dearborn Street, Chicago 10, Illinois.

Winthrop Wins Canadian Award For Medical Movie

A new medical motion picture produced by Winthrop Laboratories has won the highly-coveted Certificate of Merit of Canadian Film Awards.

The color film, entitled "Epidural Anesthesia for Vaginal Delivery in Obstetrics," was honored both for achieving its purpose and for excellent of photography. It was written by Dr. R. A. Gordon, who also handled the medical direction.

Production of the film, which was made by Chetwynd Films Limited, was under the supervision of Dr. Martin Lasersohn, executive vice-president of Winthrop.

Book Reviews

Symptom Diagnosis. By Wallace M. Yater, M.D., and William F. Oliver, M.D. Ed. 5. 951 pages. Price, $15.00. New York: Appleton-Century-Crafts, Inc., 1961.

The fact that this book is now 34 years of age and has been revised five times is evidence that it has filled a real need. The fifth edition, as the authors say, has been practically rewritten. "Although symptoms change very little, the authors were astounded at the number of new diseases described since the fourth edition appeared in 1942."

It is, of course, not possible to review this book in detail, and only a few comments will be offered. Although it contains more than a thousand pages, there are no wasted words in it. It is a model of clarity and conciseness.

The symptoms discussed are divided into regional symptoms and general symptoms. The directions "How to use this book" should be read by everyone who purchases it. A comprehensive index of 57 pages will help the reader locate readily the information wanted. A very helpful feature is an index listing first, syndromes and second, diseases, symptoms, and signs associated with men's names.

This book could have been written only by an astute clinician with an excellent command of the English language. The more familiar its owner becomes with it the more he will depend upon it. It can be heartily recommended to medical students and practitioners alike.

Sea Within: A Study of our Body Fluid. By William D. Snively, Jr., M.D. 150 pages. Philadelphia and Montreal: J. B. Lippincott Company, 1961.

Dr. Snively has written an attractive popularization of the physiology of body fluid. In a time when the television commercial has taken over adult education in things medical, it is fortunate that such a volume is available. Happily, Dr. Snively has included 55 sketches which add immeasurably to his exposition. This book can be recommended for the waiting room, for the interested layman, and will bear close scrutiny by educationists looking for a good health text for high school students.

For the person who wishes to explore more deeply, this book, of course, is no substitute for Dr. Homer Smith's **From Fish to Philosopher.** However, the tone is lighter, the illustrations are delightful, and the presentation is lucid.

Essential Pathology. By Roger D. Baker. 638 pages. Price, $9.50. Baltimore: The Williams and Wilkins Company, 1961.

With this synopsis-type volume, the Duke Department of Pathology gives a second textbook to the field, perhaps as a complement to the two-volume work of Dr. Forbus, published several years ago. The format and text follow the standard pattern of an initial section on fundamentals, "General Pathology," followed by a treatment of systemic pathology. In a small (5½" by 7½") book of this type, details must be omitted; hence the word "essential' 'in the title.

The illustrations number 470, and are of fine quality, forming the principal item in which this digest excels similar works in the field. Every pathologist reading the book would find some points of special interest to him on which he would take issue with Dr. Baker, but in general the text conforms to the commonly held opinions of the present time. For the hurried medical student, as a supplement to more complete works, or for the man in another field who wishes a short review-reference work, this volume can be recommended.

Memoirs of a Medico. By E. Martinez Alonso. 335 pages. Price, $4.50. Garden City, New York: Doubleday & Company, Inc., 1961.

Although born in Spain, with his professional career thus far in that country, Dr. Alonso was educated in Great Britain, while his father was the consul of Uruguay in Glasgow and Liverpool. The book deals with his reminiscences of growing up in a diplomatic family with 11 children; observations on the life of foreign student in Great Britain; the Spanish civil war, and the medical practice derived from his current position as doctor to the Castellana Hilton in Madrid.

The style of the book is pleasant, and it furnishes a light weekend's reading for anyone interested in the locale mentioned. The section dealing with the doctor's experiences as a surgeon, first for communist forces and later for General Franco's troops, presents an aspect of that prelude to World War II which has not received much attention in this country. Dr. Alonso claims political indifference, his duties on both sides of the conflict being explained by a death sentence passed by the Reds for alleged intrigue against their cause, followed by a fortunate escape and the gaining of a commission with the Franco forces through family connections.

To a physician in the United States an appointment as hotel physician does not appear as attractive as it does to Dr. Alonso, but he points out the reasons for its appeal to him. His anecdotes concerning the hotel guests, their behavior and their ailments are both irritating, amusing and depressing, depending on one's outlook on the segment of society likely to pass through such an hostelry. The book would make a nice bit of diversionary reading for a member of the profession of for someone with a vicarious interest in it. There must be many people in the latter category.

Cardiac Emergencies. By Harold D. Levine, M.D. 381 pages. Price, $12.00. New York: Landsberger Medical Books, 1960.

Dr. Levine relates his personal experience and that of others in the field, primarily those with whom he is associated. He provides a simple but thorough explanation of the different cardiac emergencies. Controversal points such as anticoagulants and armchair therapy in myocardial infarctions, and treatment of refractory failure are well presented. The book includes good discussions on drug intovications and arrhythmias, their recognition and therapy, as well as cardiac arrest and resuscitation.

This book provides the physician with a good, easy to read, and well indexed reference for the handling of cardiac emergencies without a lengthy explanation about the subject.

Surgical and Maxillofacial Prosthesis. By O. E. Beder. 94 pages. Price $4.00. Seattle, Washington: University of Washington Press, 1961.

This manual makes available for the first time in one volume a concise discussion of the major phases of surgical and maxillofacial or orofacial prosthesis. It was prepared hy Dr. O. E. Beder for use in his classes in the Prosthodontics Department at the University of Washington School of Dentistry. Contents include The Emotional Problems of the Occasional Patient, Splints, Stents: Their Use in Intraoral Grafts of Skin or Mucous Membrane, Protective Shields and Applicators in Radiation Therapy, Appliances for the Osteotomized or Ostectomized and Traumatized Mandible, Extraoral Impressions, and so forth.

Copies of the book are available for examination upon request from professors who wish to consider it as a text. It may be kept without charge if the publishers are notified that an order for at least ten copies has gone through. Otherwise the book may be purchased at an educational discount of 10 per cent or returned for full credit.

Winthrop Laboratories has added five new medical motion pictures to its film library and has announced that they are available immediately for professional showings.

The five new movies are 16mm. color and sound productions. Their titles are: "Avoiding Complications of Spinal Anesthesia," "Epidural Anesthesia for Vaginal Delivery in Obstetrics," "Hepatic Trauma, New Concept of Surgical Repair," "Transvaginal Regional Anesthesia in Obstetrics," and "Disinfection of the Skin."

Prints of the movies are available free for showing to medical and scientific organizations, medical students and nurses, hospitals and other professional teaching institutions. Requests for prints should be addressed to: Winthrop Laboratories, Motion Picture Department, 1450 Broadway, New York 18, New York.

In Memoriam

Joseph Alxander Elliott, M.D.
November 19, 188-May 6, 1961

Joseph Alexander Elliott was born in Moundville, Alabama, on November 19, 1888. He received his A.B. from Birmingham Southern in 1910. Having decided to devote his life to medicine, he entered the University of Michigan and received his M.D. in 1914. He chose dermatology as his specialty and served an internship in Barnod Free Skin and Cancer Hospital in St. Louis. He returned to the University of Michigan for his residency, and in 1917-1918 was appointed acting associate professor of dermatology in the Medical School. The fact that he was born and reared in the deep south, educated in his chosen profession in the far north, and received much of his training in the mid-west, undoubtedly broadened his vision and his outlook on life and prepared him for the position of leadership in his social, professional, and religious life.

Dr. Elliott came to Charlotte in 1919 as dermatologist for the Crowell Clinic, serving in that capacity until 1929, when he entered private practice.

In spite of conducting a large private practice, he always had the time, energy, and interest to advance organized medicine in his city, county, and state. He served as treasurer and later as president of the Mecklenburg County Medical Society. For many years, he was counselor of the State of North Carolina Medical Society, and served as its president in 1953. As a leader in his specialty, he was elected president of the Southeastern Dermatological Society.

It was largely through his energy and foresight that the present Doctors Building was constructed, and until ill health intervened, he served as president of the board of directors.

A biliography of some 40 published articles attests to his acuity and ability as a leader in the field of dermatology.

He took an active part in the social life of the city as a member of the Kiwanis Club, the Charlotte Country Club, and the Myers Park Country Club.

Dr. Elliott was not unmindful of his religious duties. As a member of Trinity Methodist Church when it merged to form the First Methodist Church, and later as a member of the Myers Park Methodist Church, he served on the board of stewards of each congregation.

His joy in recreation was expressed in hunting and fishing and in the culture of flowers. Always his garden was one of the show places in the city. Truly it could be said of him that he followed the biblical injunction to "do with thy might whatsoever thy hands find to do."

Acknowledging, therefore, the great contributions that Joseph Elliott made to this Society, to the profession of Medicine and to this community, be it resolved by Mecklenburg County Medical Society that a copy of this paper be recorded on the minutes of this Society as a permanent memorial to him and that a copy be sent to his family.

David G. Welton
E. J. Wannamaker
Claude B. Squires
Thomas D. Sparrow

New Device for Help in Making Cardiac Diagnoses

A prominent cardiologist reported in Philadelphia that more than half of the heart abnormalities of a group of patients he studied were detected only or mainly while the people were exercising.

Dr. Samuel Bellet, chief of the Division of Cardiology of Philadelphia General Hospital, observed 147 patients with hypertensive or arteriosclerotic cardiovascular disease and found that 58 per cent of the abnormal changes were revealed only or mainly during exercise.

Dr. Bellet's findings were presented in a scientific exhibit at the American Medical Association's Annual Meeting. Assisting him in the study which covered a total of 339 patients were Drs. Stavros Deliyiannis and Marcel Elickim of Philadelphia General Hospital.

The studies were conducted with a new diagnostic instrument, the RKG **100**, which makes practical for the first time the taking of electrocardiograms while patients are exercising.

It consists of specially designed electrodes, a pocket-sized transmitter and a desk-top receiver. The RKG **100** was developed by Telemedics, Inc., of Southampton, Pennsylvania, a subsidiary of Vector Manufacturing Company, Inc.

New Film on Auditory Screening

The Child Growth and Development Study of the Johns Hopkins University and the Maryland State Department of Health have just produced a 15 minute, 16mm., color film entitled "Auditory Screening for Infants." The purpose of the film is to stimulate interest in an auditory screening technique for infants which is not only an effective procedure for early detection of hearing impairments but which may also indicate possible abnormalities in motor coordination and mental capacity.

Preliminary plans have been made to establish training institutes at Johns Hopkins for the purpose of teaching this auditory screening technique to physicians, nurses, and other professional personnel in speech and hearing.

This film will be of interest to private physicians, nurses, health departments, medical and nursing schools, schools of public health, and speech and hearing centers.

The Month in Washington

The American Medical Association and the federal government declared all-out war on medical quacks and charlatans who milk the sick and gullible of hundreds of millions of dollars each year through useless gadgets, phony nostrums, fake reducing pills, and the many other gimmicks of the medicine show trade.

The campaign was launched at the First National Congress on Medical Quackery, under the joint sponsorship of the A.M.A. and the U.S. Food and Drug Administration, October 6-7 at the Sheraton-Park Hotel in Washington.

Among the keynote speakers were two top officials in President Kennedy's cabinet, Secretary of Health, Education and Welfare Abraham A. Ribicoff and Postmaster General J. Edward Day. Leonard W. Larson, M.D., president of the A.M.A., and Oliver Field, Director of the A.M.A. Department of Investigation, spoke for organized medicine.

Others on the program included Herbert J. Miller, assistant U.S. attorney general in charge of the criminal division; George P. Larrick, commissioner of the FDA, and Paul Rand Dixon, chairman of the Federal Trade Commission.

Other speakers included representatives of the American Cancer Society, the Arthritis and Rheumatism Foundation, and the National Better Business Bureau.

C. Joseph Stetler, director of the Legal and Socio-Economic Division of the A.M.A. presided at the meeting.

Many state and county medical societies from throughout the nation sent representatives to the Congress. They carried back to their societies plans for cooperation with enforcement agencies at the local level and for a step-up of public education on the subject in an accelerated campaign against quacks.

Highlights of the talks included:

—Larson: "We must educate the public thoroughly and effectively. We must wage psychological as well as scientific warfare. We must not only prove the worthlessness of quackery, but we also must establish confidence in sound medical and health care.

"Speaking for the American Medical Association and our 180,000 physician-members, I pledge our efforts to the final eradication of quackery and all its minions and satraps."

—Ribicoff: "The total cost of unnecessary or dangerous medications in this country probably exceeds $1 billion each year. Much of this expense is to men, women, and children who dearly need this money for good medical care or for other necessities of life.

"But quackery's costs in dollars only introduces the story. In terms of false hopes raised, in terms of ugly delusions fostered, in terms of tinkering with human life itself, the cost cannot be measured. The quack flirts with disaster. He challenges the sixth Commandment: 'Thou shalt not kill.' "

—Larrick: "The most widespread and expensive type of quackery in the United States today is in the promotion of vitamin products, special dietary foods, and food supplements. Millions of consumers are being misled concerning their need for such products. Complicating this problem is a vast and growing 'folk-lore' or 'mythology' of nutrition which is being built up by pseudo-scientific literature in books, pamphlets, and periodicals. As a result, millions of people are attempting self-medication for imaginary and real illnesses with a multiude of more or less irrational food items. Food quackery today can only be compared to the patent medicine craze which reached its height in the last century. Especially disturbing is the tendency shown by some big and hitherto respected food concerns to use quackery in their sales material."

—Dixon: "Properly drafted and administered, legislation giving the Federal Trade Commission power to issue temporary cease-and-desist orders would, while observing all the requirements of due process, make it possible to protect the public interest more adequately in many areas.

"Although in the case of food, drug, and cosmetic advertising, the Commission can . . . apply to district courts for temporary

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From the Washington Office of the American Medical Association.

injunctions, it would be much more efficient for the Commission itself to issue temporary orders in those cases as well as in others."

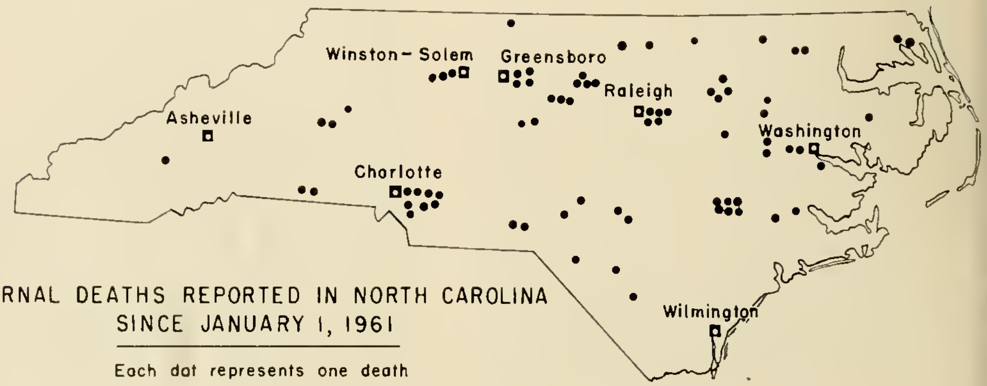
—Day: "The peddling of fake medical cures is the most prominent fraudulent activity conducted through the U.S. mails today. This huge 'industry'—and it has grown to that extent—is so prevalent and so widespread that it taxes the manpower of the Postal Inspection Service to the utmost in trying to bring the perpetrators to justice.

"We are doing everything we can to make more of our inspectors available to work on cases of this nature, to the extent it will not jeopardize enforcement in other fields."

Dr. L. Henry Garland, American Cancer Society: "The charlatan is in business to make money and he does so by offering *hope*. He tends to be courteous, optimistic, easily understood by the laymen, and confident that cure can be obtained. His patient does not care that the method used is a secret one, that the testimonials are largely fraudulent, or that the 'doctor' may not even be licensed. All he knows is that he is being reassured and treated by someone who seems to be interested in him as a person."

—Dr. R. W. Lamont-Havers, Arthritis and Rheumatism Foundation: "That this is a large problem is indicated by the estimated 250 million dollars a year that arthritic victims spend upon unproven, and misrepresented products in a vain attempt to obtain unrealizable relief from their suffering. Not all of these products are quackery in the sense of being useless. Some contain active ingredients—usually salicylates, or apparatus such as vibrators, but are promoted with such misrepresentation of effects that the arthritic fully expects results beyond the capabilities of the drug. Others are outright quackery and include such popular items as alfalfa tea, uranium pads, honey and vinegar, etc. Of particular concern are the widely advertised so-called 'clinics,' chiefly in Missouri and Florida."

—Field: "We would like to envision the time when we can cease to worry about the medical quack. But it's going to take an awful lot of doing. The Food and Drug Administration, the Post Office Department, the Federal Trade Commission and the Food and Drug groups of many states of the Union, cannot do the job alone. It takes a program which seeks to acquaint the public with the problem, and swings into action quickly when there is a threat to the community or to the nation at large. This takes the help of all interested people—consumer groups, educational groups, religious organizations, and, most of all, those responsible for the education of the American youth . . . The emphasis should be on letting the public know, strengthening the laws where necessary, but, most of all, providing a means of distinguishing between the legitimate medical practitioner and the one who pretends to be one."



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Each dot represents one death

NORTH CAROLINA Medical Journal

MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

December, 1961
Vol. 22 No. 12

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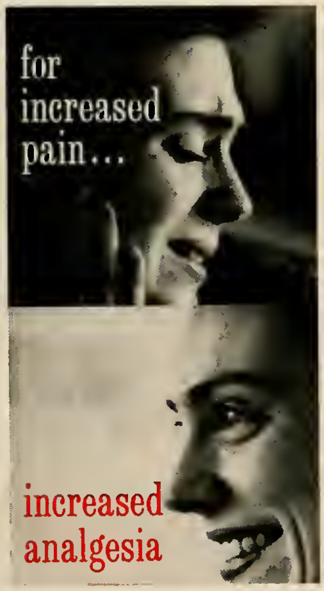

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- ✓ Adsorbs toxins and gases
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(equivalent to 2 cc. paregoric)

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NORTH CAROLINA MEDICAL JOURNAL

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VOLUME 22 DECEMBER, 1961 NUMBER 12

The Physician's Reaction to the Patient with Functional Illness

JOHN B. RECKLESS, M.B., Ch.B.
DURHAM

It has been estimated that 60 per cent of the patients who consult a physician do so because of conditions which are partly emotional in origin. In many of the remaining 40 per cent of patients with organic illnesses, there is an emotional component which must be taken into account in the treatment process[1].

Because psychiatric treatment is usually expensive and sometimes difficult to obtain, the general practitioner and internist find that they must cope with a considerable number of patients whose symptoms have an emotional basis. Many physicians are reluctant to treat such patients—partly because the way in which the patient with a functional illness presents himself often arouses negative feelings in the physician. With a better understanding of his own reaction to such patients, and an improved technique for interviewing them, the general practitioner or internist will be far more effective in the management of functional illnesses.

Characteristics of Patients With Functional Illness

As here defined, the term *functional* applies not only to physical symptoms produced by emotional reaction but also to organic illnesses which are influenced by unrecognized emotional components.

Certain annoying traits, although present in varying degrees, are characteristic of patients with functional illness.

1. Such patients present a confused and rambling account of their symptoms and complaints.
2. They continually shift their emphasis. As fast as one set of complaints is investigated, other complaints in other systems emerge.
3. They make unusual demands on the physician's time and energies. Suggestions and instructions are not consistently followed.
4. Sincere attempts to help often seem to be misunderstood, and the patient may appear to have little desire to get well.
5. Treatment often seems ineffective, and the complaints tend to chronicity.
6. No clear-cut cause for their symptoms can be uncovered.

Recognizing Negative Feelings Toward a Patient

With such a pattern, it is little wonder that we physicians tend to have negative feelings about these patients[2]. It is important that we should recognize our feelings, for they will affect our behavior toward the patient. Some patients, of course, arouse over-positive feelings, and these too will distort our judgment concerning diagnosis and treatment. By practicing the habit of self-observation, we will discover that a state of neutrality toward a patient seldom

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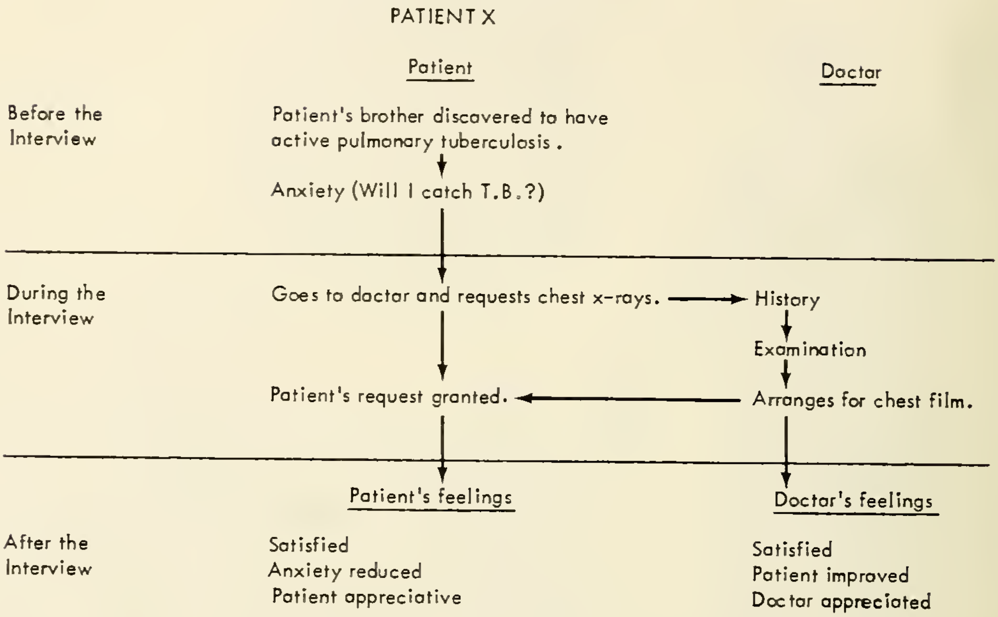
Read (under the title "Are You Bothered by the Nervous Patient?") before the Section on General Practice, Medical Society of the State of North Carolina, Asheville, May 9, 1961.

Reread upon request at the Utah State Medical Association Meeting, Salt Lake City, Utah, September 15, 1961.

From the Department of Psychiatry, Duke University Medical Center, Durham, N. C.

The Doctor-Patient Interactional Chart



exists, and that our emotions influence us throughout all of our waking hours. Our reaction to the same person or situation will vary from time to time, depending on how busy we are, the state of our health, our personal problems, and our state of knowledge regarding the problem at hand.

Start taking note of the changes in your attitude as you encounter patients of different age, sex, social standing, and degree of acquaintance. Before a particular patient arrives, do you anticipate the contact with foreboding or with pleasure? As you glance at the clock when he enters the office, do you feel glad that there is plenty of time, or irritated that you have no excuse to get away from him in a hurry? Are there certain patients whose histories you can't remember, or whom you forget to visit or phone or write a letter for? Such memory lapses may indicate the presence of negative feelings toward the patient.

By observing our reactions to different types of patients, we may be able to anticipate our reaction to a particular patient, and thus may be able to avoid error or bias resulting from our interview with him.

Interviewing the Patient with Functional Illness

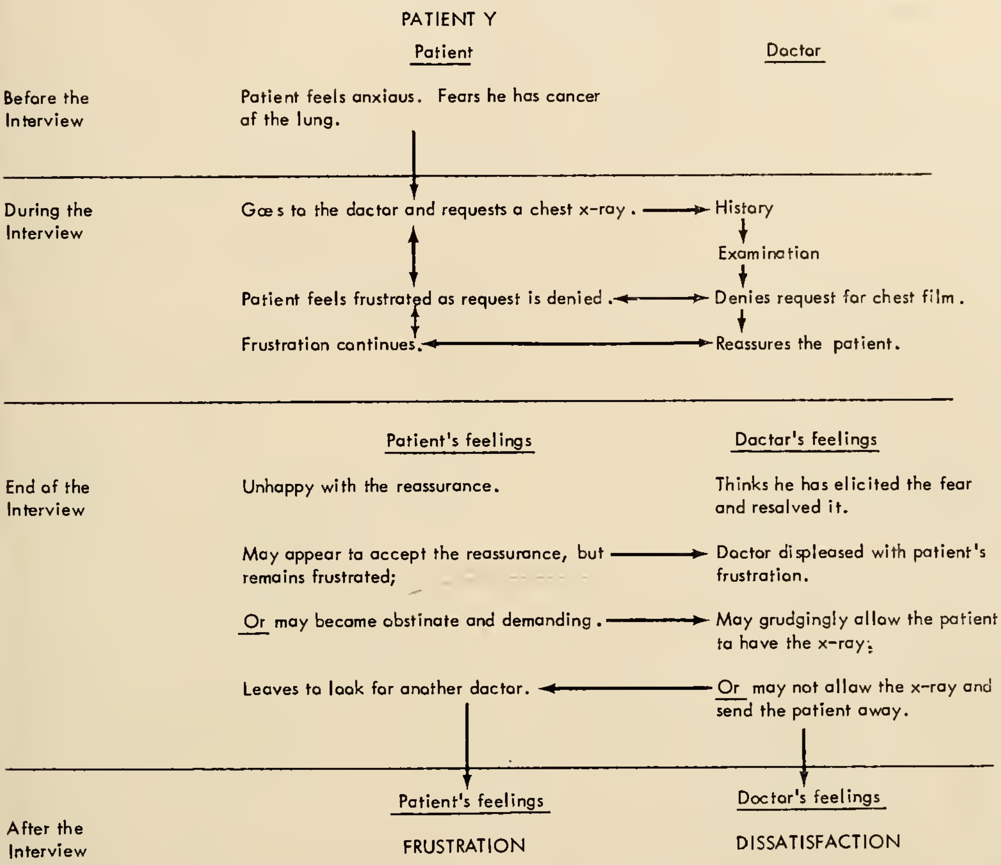
The opening interview with a patient who has a functional illness is extremely important. The usual question-and-answer method of history-taking somehow falls down, and leaves the physician with the feeling that he does not really understand the meaning of the patient's physical complaints.

Let us consider two hypothetical cases in which the standard interview technique might be employed, with different results.

Patient X came to a physician's office and requested an x-ray of the chest. On questioning him, the doctor learned that he had been living with a brother who had been found to have active pulmonary tuberculosis. He considered the patient's request reasonable, and arranged for the chest film to be made.

Patient Y, on the other hand, seemed nervous and ill at ease when he entered the office. He began by stating that he wanted an x-ray of the chest, then told of a tightness in his chest and confessed that he was afraid he had cancer. Although the doctor thought that the patient's complaints were

The Usual Type of Interview


PATIENT Y
Patient
Doctor
Before the Interview
Patient feels anxious. Fears he has cancer of the lung.
During the Interview
Goes to the doctor and requests a chest x-ray.
History
Examination
Patient feels frustrated as request is denied.
Denies request for chest film.
Frustration continues.
Reassures the patient.
Patient's feelings
Doctor's feelings
End of the Interview
Unhappy with the reassurance.
Thinks he has elicited the fear and resolved it.
May appear to accept the reassurance, but remains frustrated;
Doctor displeased with patient's frustration.
Or may become obstinate and demanding.
May grudgingly allow the patient to have the x-ray;
Leaves to look for another doctor.
Or may not allow the x-ray and send the patient away.
After the Interview
Patient's feelings
FRUSTRATION
Doctor's feelings
DISSATISFACTION

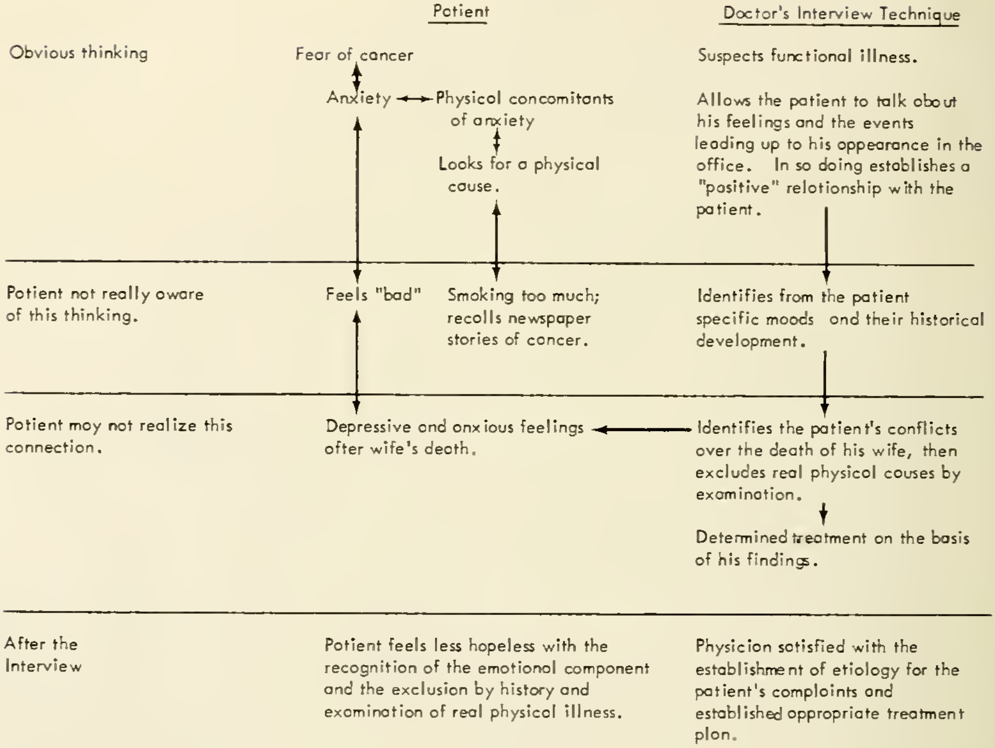
probably functional, he took a thorough history and performed a complete examination. He then told the patient that there was no evidence of cancer; that his heart, lungs, and general physical condition were sound; and that there was no need for him to worry or go to the expense of having a chest film made.

This hypothetical patient's reaction to such a situation would depend on the patient's image of the doctor and the strength of his feeling that he must have an x-ray. He might appear to accept the doctor's reassurance, thank him, go away, and promptly seek out another physician; or he might argue and persist in his request for an x-ray. The physician, in turn, might accede to his request and arrange for the x-ray, or he might become obstinate and refuse to do so. In either case, he would in all probability have very definite feelings concerning this patient, and these might range from despair to frank irritation and annoyance. Certainly he would remember those feelings if the patient ever called for another appointment.

How different would have been the result had the doctor allowed the patient to describe what feeling lay behind his request for the x-ray! The patient was clearly under stress. He felt uneasy and anxious, and had vague aches and pains in the body, with a sense of tightness in the chest. He was afraid that the doctor might laugh at him if he said he had a tight feeling in his chest. These physical symptoms had started after his wife's recent death, but he didn't suppose there was any connection. He had never been ill before. He decided that he

The Recommended Interview Pattern

PATIENT Y



	Patient	Doctor's Interview Technique
Obvious thinking	Fear of cancer Anxiety ⟷ Physical concomitants of anxiety Looks for a physical cause.	Suspects functional illness. Allows the patient to talk about his feelings and the events leading up to his appearance in the office. In so doing establishes a "positive" relationship with the patient.
Patient not really aware of this thinking.	Feels "bad" Smoking too much; recalls newspaper stories of cancer.	Identifies from the patient specific moods and their historical development.
Patient may not realize this connection.	Depressive and anxious feelings after wife's death.	Identifies the patient's conflicts over the death of his wife, then excludes real physical causes by examination. Determined treatment on the basis of his findings.
After the Interview	Patient feels less hopeless with the recognition of the emotional component and the exclusion by history and examination of real physical illness.	Physician satisfied with the establishment of etiology for the patient's complaints and established appropriate treatment plan.

had better see a doctor; perhaps *he* could find out what was the matter with him. "That's it! I'll get examined and have an x-ray. Hey! How about all those newspaper reports on smoking and cancer of the lungs? I've been smoking more heavily recently. Yes. *I must see a doctor and get an x-ray, for I may have cancer.*"[3]

How did the physician discover the emotional factors responsible for this patient's symptoms and his fear of lung cancer? Here are some specific suggestions for interviewing such patients in order to uncover the emotional components of their illness:

1. Allow more time for dealing with such patients.
2. Keep a receptive mind during the interview.
3. *Stop asking questions and allow the patient to tell the history in his own language.* When the patient feels that the doctor is sympathetic and concerned, and is not passing judgment on him, he is likely to tell the doctor far more about his real problems and less about his symptoms. At the end of the interview, however, he may again attempt to blame his symptoms on some organic disease.
4. Listen to the patient's *words*: "My stomach *growls*," or "My bowels feel *tight*." Both "growl" and "tight" suggest a mood—anger and fear, respectively. Note also the moment of hesitation and the silence, laughter, or tears which reveal the patient's emotional reaction to a particular phase of his history.
5. Note the *order* of complaints; the last one may be the one that the patient is really concerned about.
6. Don't accept at face value the patient's first explanation of his symptoms, no

matter how rational it seems. An example is the time-worn phrase, "I've been working too hard recently." This is seldom the real explanation of the patient's difficulty.

7. Listen carefully to the patient's opening and closing remarks; they are often a camouflage for the patient's real fears, and by probing for what lies behind them the doctor can sometimes uncover the hidden emotional problem. Mr. Y's request for an x-ray is an example of an opening camouflage. Even more important than the patient's opening remarks, however, are his final words as he leaves the office. The phrase, "Well, doctor, you don't think I am going to die after all," may express the patient's real fear, which he did not disclose earlier for fear of being laughed at. The remark is made jokingly at the end of the interview, and the patient makes a quick exit if he perceives that the doctor thinks his question is foolish. It is unfortunate that at this moment, when the patient is most ready to talk, the doctor is least ready to listen. If he would call the patient back and suggest that they look into that phrase, he might be able to get to the root of the patient's anxiety.

8. *DON'T*
   a. Give too much advice.
   b. Tear down the patient's defenses by tell him flatly, "There's nothing organically wrong with you." Leave him his self-respect by letting him know that you realize his symptoms have some explanation, even though you have not been able to uncover it.
   c. Jump to conclusions regarding the cause of seemingly functional complaints. A patient with obvious emotional problems may have other fears and anxieties which are less obvious but more basic; or he may have an obscure physical disease which masquerades as a functional disorder[4]. Anxiety may occur with hyperthyroidism or a pheochromocytoma, a neurasthenic syndrome with chronic adrenal disease or anemia, depression with arteriosclerotic brain disease or carcinoma, and a schizophrenic pattern with a brain tumor or myxedema; mood swings are characteristic of systemic tuberculosis, and general paralysis of the insane has been called the great imitator.

How the Physician Can Overcome His Negative Feelings Toward Patients With Functional Illness

I mentioned earlier that the physician's feeling of inadequacy when confronted with a patient whose problem lies largely in the emotional realm might depend in part on the state of his knowledge concerning that patient's problem. The general practitioner or internist who is interested in psychosomatic medicine and up-to-date in his knowledge of the subject is usually challenged rather than appalled by the appearance of a patient with functional illness. Fortunately, postgraduate courses in psychiatric subjects are now being made available in North Carolina and other southern states through the activities and financial support of the Southern Regional Education Board[1] and a variety of federal government grants. These courses deal with such subjects as psychiatric symptomatology and its causes; the recognition of depressive states and appropriate drug therapy (for not all antidepressants have an equal effectiveness in all types of depressive illness[5]); techniques of interviewing, and short-term factors in psychosomatic disease.

As a result of the improved psychiatric curriculum in the medical schools, future medical graduates should be better equipped to deal with functional illness. At Duke, as at many other medical schools, the undergraduate student spends 400 hours in the psychiatric program, learning skills which he is encouraged to use in other fields of medicine, particularly in internal medicine.

Finally, the general practitioner or internist will feel less negative toward patients with functional illness if he has a cordial professional relationship with at least one psychiatrist, whom he can call on for help when he has exahusted his resources with a particular patient[6].

Summary

One of the chief sources of difficulty for the general practitioner in treating patients with functional and emotional disorders is the negative feelings which such patients often arouse in the physician. An undertaking of this doctor-patient interaction, together with improved interview techniques, can make the treatment of patients with functional illness far more effective. Postgraduate courses are available to help the general practitioner increase his knowledge of functional and emotional illnesses.

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Radiologic Findings in Primary Carcinoma of the Gallbladder

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Carcinoma of the gallbladder is not as rare as the numerous reports of relatively small series of cases might suggest. It has been estimated that more than 6,000 persons in the United States die of it each year[1]. Nearly 5,000 cases of primary carcinoma of the gallbladder have been documented in the world literature since the first description of a case by Maximillian de Stoll in 1777[2]. The disease accounts for 8 to 10 per cent of deaths from cancer in women[3]. The American literature has been comprehensively reviewed by Arminski, Rivkin, and more recently by Strauch[4].

Despite recent advances in medical knowledge and radiologic diagnostic techniques, little progress has been made in the early clinical diagnosis of primary carcinoma of the gallbladder. This report is a resume of 15 verified cases of primary cancer of the gallbladder which were seen at the North Carolina Memorial Hospital between 1953 and January, 1961. All cases were histologically proven, and cases where the tumor was of undetermined origin were excluded. Roentgenologic examinations relative to the abdomen or digestive tract were available in 13 patients. These have been reviewed for possible diagnostic characteristics which might lead to an earlier or more definitive preoperative diagnosis of gallbladder carcinoma.

Incidence

The incidence of primary carcinoma of the gallbladder in all biliary tract operations was 1.4 per cent in Strauch's collective review, but this figure increases to 10 per cent for biliary operations in the seventh decade of life[5]. At Memorial Hospital during the period covered, primary carcinoma of the liver was encountered nine times, carcinoma of the bile ducts twice, carcinoma of the ampulla of Vater five times, and carcinoma of the pancreas 74 times.

Carcinoma of the gallbladder is a disease of the "cancer age," the great majority of patients being in the sixth and seventh decades of life. Females are afflicted three times as often as males, a preponderance which has long been observed in calculous and other benign types of gallbladder dis-

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eases. In the Memorial Hospital group, the average age on admission was 61 years, the youngest patient being a 41 year old male and the eldest a centenarian female who was paying her first visit to a hospital. Eleven, or 73 per cent, of the patients were females.

Histology

The histology of primary malignancy of the gallbladder has been amply described[6]. More than 85 per cent of primary malignant tumors of the gallbladder are adenocarcinomas of the scirrhous, papillary, or mucin-producing types. Epidermoid carcinomas, undifferentiated carcinomas, and adenoacanthomas comprise the majority of the remaining tumors. Fourteen cases in our group were of the adenocarcinoma variety, and one tumor was an epidermoid carcinoma.

Clinical Findings

The clinical picture associated with gallbladder cancer is variable. Symptoms and signs may be indistinguishable from those of acute or chronic benign biliary tract disease. More commonly the picture is that of a progressive neoplastic process superimposed on chronic biliary symptoms of many years' duration. Occasionally, presenting symptoms are those arising from involvement of surrounding organs, and overshadow the primary lesion in the gallbladder. Eleven patients in our series had a past history of chronic gallbladder disease ranging from 5 to 50 years' duration. Pain, usually localized in the right upper quadrant or epigastrium, was the most common symptom, being present in 80 per cent of the cases, and was frequently the presenting complaint. One patient with spinal metastases complained of upper back and chest pain. Nausea and vomiting was a major complaint in 3 patients and was an associated symptom in 6 others. Two patients had symptoms consistent with upper intestinal obstruction. Four-fifths of the group had a history of recent significant weight loss. Gastrointestinal hemorrhage was observed in 3 cases, 2 of which had episodes of massive hematemesis requiring whole blood transfusions.

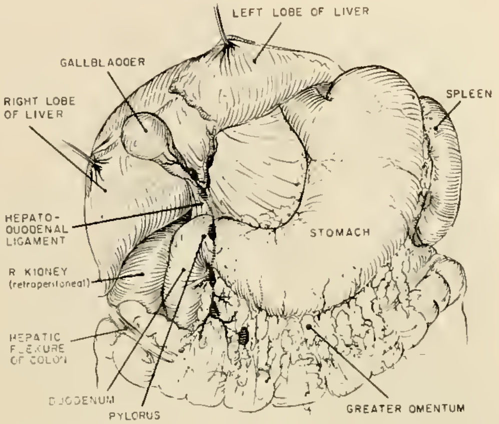

Fig. 1. Anatomy and lymphatic drainage of the gallbladder.

Six of the 15 patients were icteric on admission to the hospital, and a seventh exhibited an elevated serum bilirubin, although clinical signs of jaundice were not evident. The jaundice was found to be of the obstructive variety by laboratory tests, and the serum alkaline phosphatase was usually elevated.

The most consistent finding on physical examination was a palpable mass in the right upper quadrant. This finding was present in more than one half of the patients. Clinical hepatomegaly was frequently present, although it was not always possible to distinguish the inferior border of the liver when there was an associated extrahepatic mass in the right side of the abdomen. Nevertheless, liver metastases were subsequently confirmed at laparotomy in 13 of 14 patients where the organ could be adequately examined.

Roentgenologic Findings

A review of the anatomy of the right upper quadrant and the lymphatic drainage of the gallbladder[7] is helpful in understanding the gross pathologic picture and roentgenographic findings associated with cholecystic cancer (fig. 1). The gallbladder is a subserosal sac which lies in intimate contact with the inferior surface of the liver. Its fundus frequently extends beyond the anterior liver edge to lie in contact with

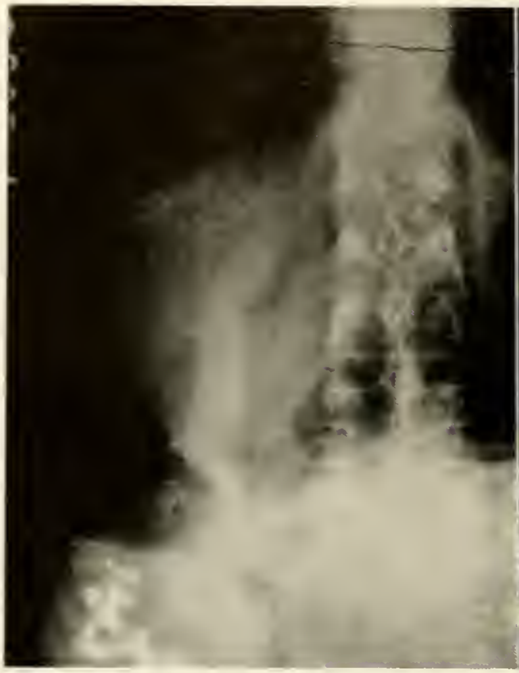

Fig. 2. Carcinoma of gallbladder (fundus) with concentration of radiopaque contrast media in the organ. This is a rare occurrence in cancer of the gallbladder.

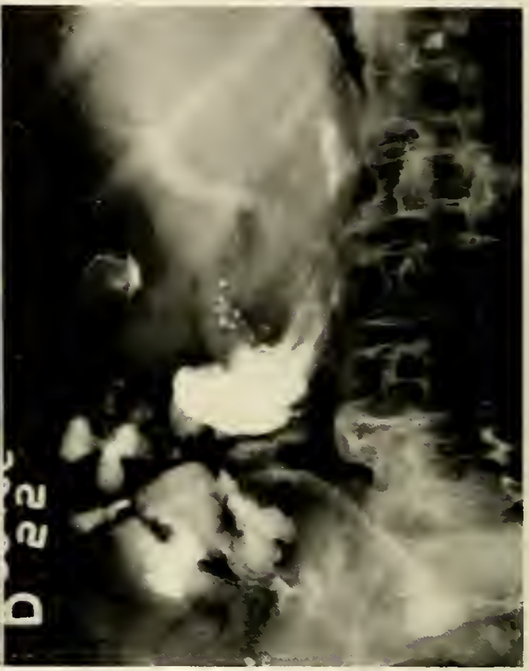

Fig. 3. Intravenous cholangiogram demonstrating the common bile duct. The gallbladder lumen did not opacify.

the abdominal wall or superior border of the transverse colon. Its posterior surface is closely related to the ventral surface of the right kidney and the anterolateral border of the proximal portion of the duodenal loop.

The submucosal and subserosal lymphatic plexuses of the gallbladder have numerous paths of intercommunication through the muscular layers, the latter plexus forming several main channels which follow the cystic artery to empty into nodes at the neck of the organ. From there, the lymphatics join a chain of nodes in the hepatoduodenal ligament which communicate with the hilus of the liver superiorly and the peripancreatic and celiac nodes inferiorly. There are also intimate connections between the lymphatics of the liver and those of the subserosal layer of the gallbladder. Regional lymphatics showed gross or microscopic evidence of metastatic tumor in each of 14 patients in our series who underwent laparotomy. The presence of tumor masses along these lymphatic pathways was responsible for some of the most profound roentgenologic changes observed in association with the disease.

Previous reports of cancer of the gallbladder have emphasized largely the absence of gallbladder opacification at cholecystography, together with the association of calculi, when radiopaque, as the only consistent radiographic findings. Since these changes are much more frequently manifestations of benign biliary tract disease, they have not been of much help in the diagnosis of malignancy. Of 10 patients who had one or more cholecystographic studies either by the oral or intravenous technique, only one exhibited significant concentration of the radiopaque media by the gallbladder (fig. 2). This patient, a 100 year old woman with no symptoms of biliary disease, had an adenocarcinoma in the fundus of the gallbladder that had invaded the anterior abdominal wall. An incisional biopsy of the lesion resulted in a cholecysto-cutaneous fistula. Poor or non-opacification is the rule with tumors involving the infundibulum or in organs with pre-existing cholecystitis and cholelithiasis.

The rôle of intravenous cholangiography and cholecystography in the diagnosis of primary carcinoma of the gallbladder has not been stressed in previous reports. Two cases studied by this method demonstrated normal common ducts but unsatisfactory opacification of the gallbladder lumen (fig. 3). The technique might prove helpful in differentiating tumors of the ampulla of Vater and head of the pancreas from primary carcinoma of the gallbladder, but more experience is needed.

Calculi associated with carcinoma are frequently non-radiopaque, and cannot be demonstrated radiographically without contrast opacification of the gallbladder lumen. Donhauser[8] reported no calculi visualized in 30 of 33 patients in whom calculi were present. Review of abdominal films in our 4 cases with surgically proved calculi failed to yield evidence of positive shadows. In 2 cases, intramural gallbladder calcifications were radiologically demonstrable (figs. 4 and 5). In one case this was manifested as a fine stippling throughout the gallbladder wall, and in the other as a more dense eggshell type of deposit localized in the fundus.

Contrast examination of the upper gastrointestinal tract probably furnishes the most distinctive roentgenologic findings in primary cancer of the gallbladder. Localized pressure effects in the pyloric antrum from a large primary mass in the gallbladder, and compression of the proximal duodenal loop by metastatic nodules in the porta hepatis, have been described by McConnell[9].

Ten patients in the Memorial Hospital group underwent preoperative examinations of the upper gastrointestinal tract, 7 patients exhibiting some abnormality of the stomach or duodenum which could be attributed directly or indirectly to the primary disease process. The most characteristic deformity was an extrinsic effect along the greater curvature of the pre-pyloric an-

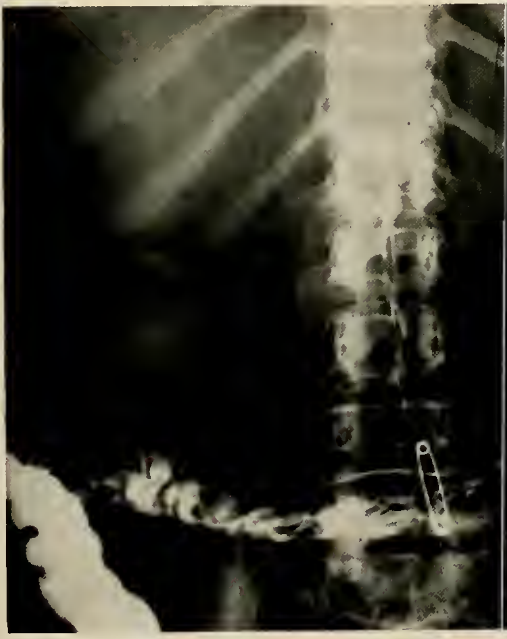

Fig. 4. Calcification in the wall of a carcinomatous gallbladder.

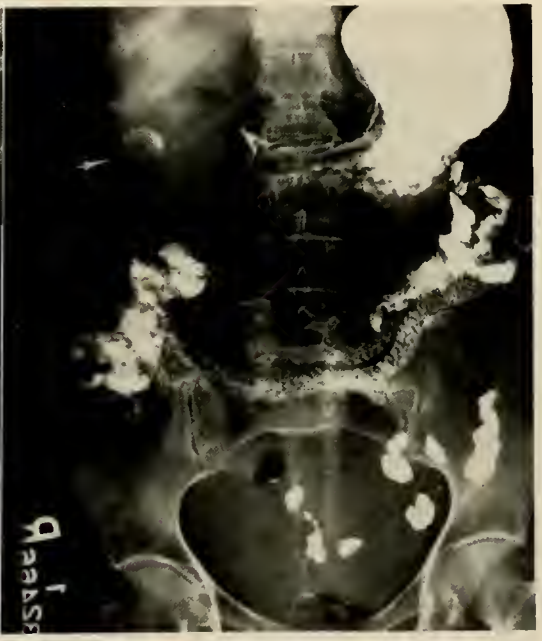

Fig. 5. Dense intramural calcification in adenocarcinoma of the gallbladder.

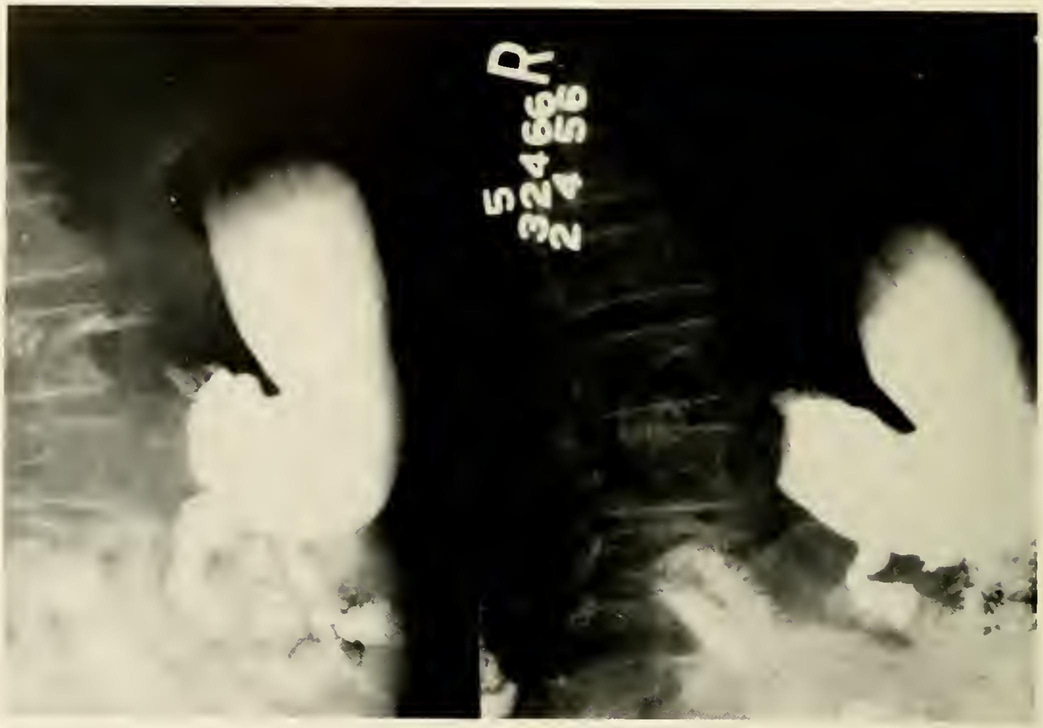

Fig. 6. Deformity of the gastric antrum and duodenum from carcinoma of the gallbladder and its regional metastases.

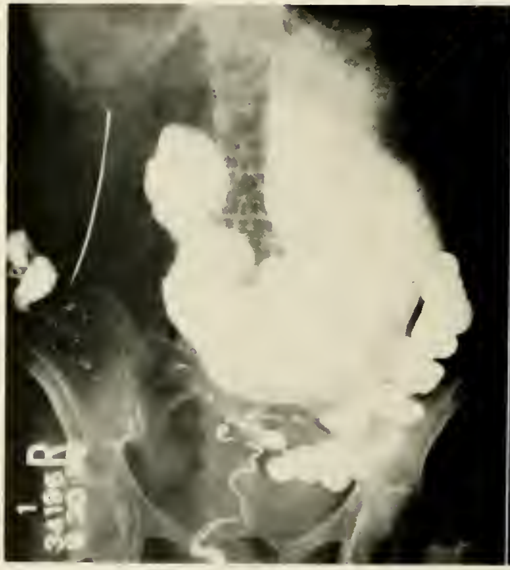

Fig. 7. Duodenal obstruction secondary to regional metastases from carcinoma of the gallbladder.

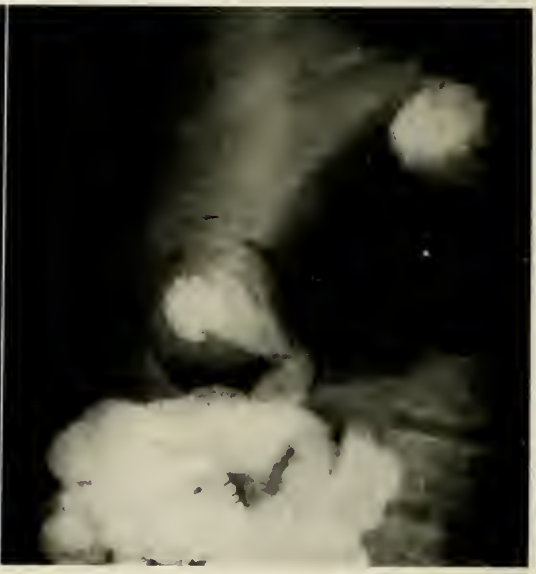

Fig. 8. Cholecystoduodenal fistula, with reflux of barium into cystic and hepatic biliary ducts.

trum and medial border of the descending duodenal segment (fig. 6). This type of deformity was usually due to compression or serosal invasion of the gastric antrum from a large primary mass in the gallbladder, in addition to extensive involvement of nodes in the hepatoduodenal ligament and peripancreatic plexus. Duodenal displacement and compression produced by lymphatic spread in the porta hepatis was seen frequently, often simulating deformity from a primary lesion arising in the head of the pancreas. Duodenal obstruction due to metastases was also encountered (fig. 7). A cholecystoduodenal fistula was demonstrated in one case (fig. 8), and is a sign which has been reported to weigh heavily in favor of carcinoma of the gallbladder[10].

Involvement of other surrounding organs was demonstrated roentgenologically in 2 cases. A submucosal defect representing involvement of the hepatic flexure of the colon (fig. 9) and distortion of the renal outline from invasion of the right kidney (fig. 10) were demonstrated in one patient. Another patient presented complete obstruction of the transverse colon from recurrent carcinoma four years after cholecystectomy. Peritoneal metastases, usually in the greater omentum, were described at operation in 4 cases. Intra-abdominal carcinomatosis is usually the final stage of the disease.

Extra-abdominal metastases from carcinoma of the gallbladder are uncommon. This was observed twice in our 15 cases. One patient presented destructive lesions in the fourth and fifth thoracic vertebrae (fig. 11), and an asymptomatic mass in the right upper quadrant was discovered on physical examination. The histologic pattern of the osseous lesion was identical to that of the primary adenocarcinoma in the gallbladder. The second patient was a 70 year old woman with diffuse nodular pulmonary metastases (fig. 12).

Diagnosis

The early clinical diagnosis of carcinoma of the gallbladder, when the disease is still confined to the primary organ and potentially curable, is difficult to conceive. Although the diagnosis of benign tumors of the gallbladder by cholecystography has been emphasized by others[11], carcinomatous transformation of these lesions is apparently uncommon[12]. The problem appears to be

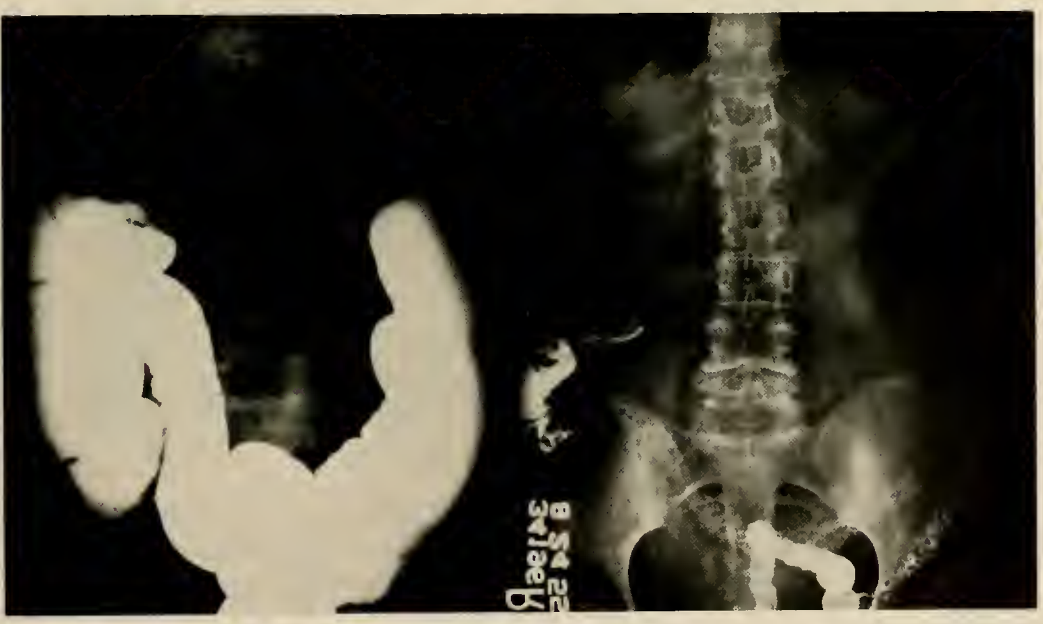

Fig. 9. Direct extension of carcinoma of the gallbladder into wall of the hepatic flexure of the colon.

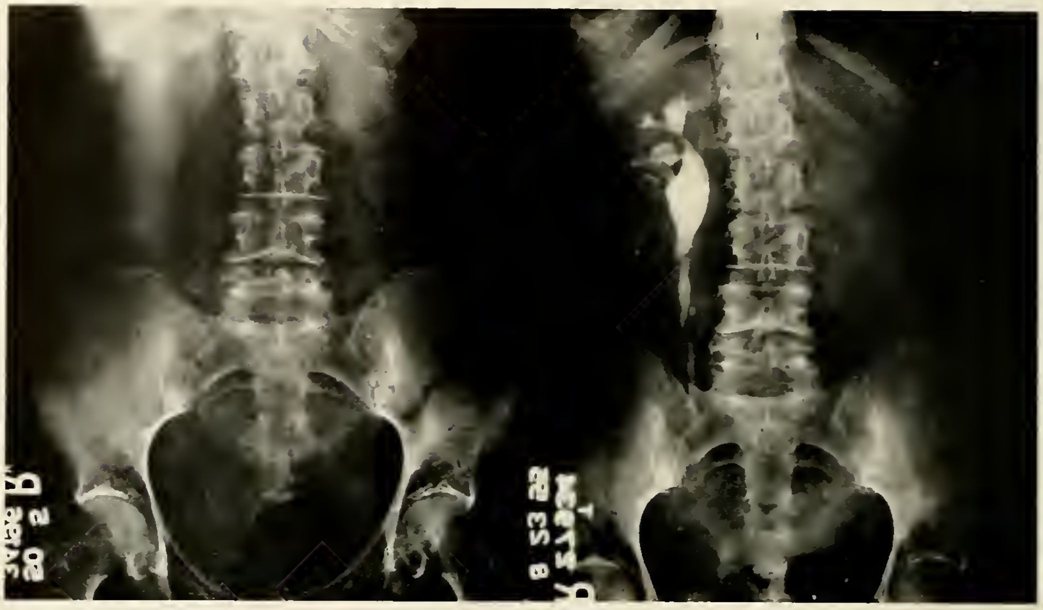

Fig. 10. Invasion of the lower pole of the right kidney by gallbladder cancer (same patient as fig. 9).

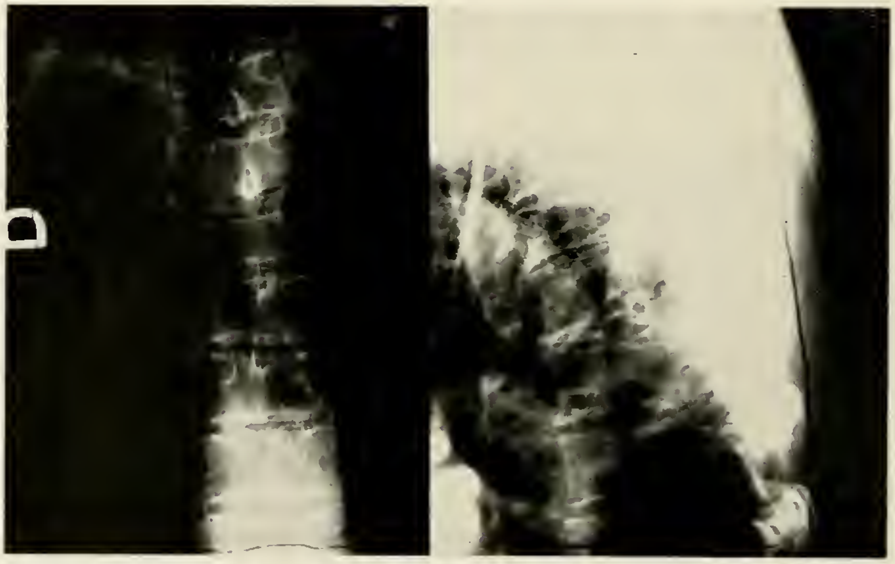

Fig. 11. Metastases to thoracic spine with destruction of pedicles of the fourth and fifth vertebrae. The primary tumor in the gallbladder was clinically silent.

more that of the early diagnosis of cholecystitis and cholelithiasis. Although calculi are found in 85 to 90 per cent of cancerous gallbladders, the incidence of carcinoma in patients with pre-existing cholelithiasis ranges from 3 to 6 per cent, according to Strauch. This incidence does not justify prophylactic surgery in every patient with asymptomatic gallstones, although the risk of developing carcinoma of the gallbladder

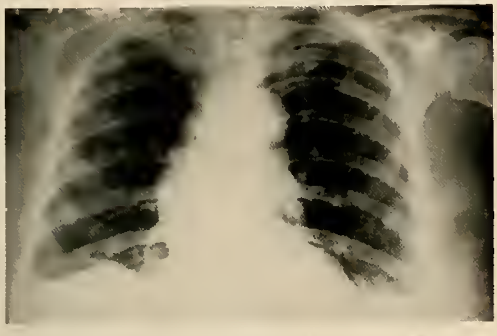

Fig. 12. Nodular pulmonary metastases from carcinoma of the gallbladder.

is four to five times greater than the operative mortality for elective cholecystectomy. The greater incidence of non-cancerous complications, such as acute cholecystitis, cholecodolithiasis, perforation of the gallbladder and pancreatitis, would seem to be a much stronger argument for prophylactic cholecystectomy in cholelithiasis.

Once the disease is well established, a preoperative diagnosis of carcinoma of the gallbladder can frequently be suggested by the radiologist. The patient is usually an elderly woman with jaundice, upper abdominal pain, a mass in the right upper quadrant, and a history of recent weight loss. X-ray studies may reveal calculous or interstitial calcifications in the region of the gallbladder, non-opacification on cholecystography, localized defects in the barium-filled stomach and duodenum, and, occasionally, fistulous communications between the gallbladder and duodenum. In some cases there will be signs of involvement of the transverse colon or right kidney.

In our 15 cases, the diagnosis of primary carcinoma of the gallbladder was made preoperatively in 4 cases and was mentioned in the differential diagnosis in 4 additional patients. Carcinoma of the head of the pancreas is the disease most frequently confused with cholecystic cancer roentgenologically. McConnell has emphasized the more anterior-superior location of gastroduodenal deformities in the latter, whereas pancreatic lesions tend to deform the stomach and duodenum more from the posterior aspect. The differentiation of carcinoma of the gallbladder from primary tumors arising in the stomach or transverse colon may occasionally be troublesome.

Treatment

It is beyond the scope of this discussion to review the various methods of treatment of gallbladder cancer. The collective five-year survival rate of 2.1 per cent[13] would indicate that, to date, the disease is remarkably resistant to all forms of therapy. There are no five-year survivors in our 15 cases. The onset of characteristic symptoms or signs of the disease usually means that widespread extension and metastases are present. In 1924 Blalock[14] stated: "In malignancy of the gallbladder, when a diagnosis can be made without exploration, no operation should be performed, inasmuch as it only shortens the patient's life." This concept does not appear to have been seriously challenged by the results of present day radical surgical techniques.

If the diagnosis of primary carcinoma of the gallbladder can be established by the correlation of clinical, laboratory and radiographic findings, and confirmed by needle biopsy of the liver, the disease is probably incurable and radical surgery contraindicated. Palliative operations for relief of intestinal obstruction, jaundice, or perforation may be required when these complications are present. Palliative radiotherapy may be justified in some cases of squamous-cell carcinoma. Recently, short-term beneficial effects have been reported with the intravenous administration of radioactive rose bengal in biliary cancer[15].

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Long-term Clinical Studies with a New Constipating Drug, Diphenoxylate Hydrochloride*

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The control of acute or chronic diarrhea resulting from functional or organic disease is a common and difficult problem of medical management. Kaolin, pectate, and bismuth preparations are of limited efficacy and value. Potent narcotics such as tincture of opium, morphine, and codeine are undesirable, particularly in chronic disorders, because of the hazard of addiction. A constipating drug which can be prescribed in tablet form, has little analgesic effect, and is not habit forming would be a valuable aid in the management of chronic diarrhea.

Pharmacology

Jansen and Jaganeau[1], following a study of the constipating and analgesic activities of various synthetic compounds, reported that diphenoxylate hydrochloride is a potent antidiarrheal and constipating agent which possesses minimal analgesic effect and no parasympatholytic activity. In mice, this drug was more effective than papaverine hydrochloride, codeine phosphate, atropine sulfate, or morphine hydrochloride in slowing the propulsion of a "charcoal meal" through the intestine. The compound's chemical relationship to meperidine, as well as roentgen studies of intestinal motility, suggests that the drug probably acts by increasing tone and decreasing intestinal peristalsis. Clinically effective doses do not delay gastric emptying.

Pharmacologic studies on human subjects, undertaken because of the drug's structural relationship to meperidine, have indicated that Lomotil is capable of preventing withdrawal symptoms in known narcotic addicts, although it apparently produces neither dependency nor analgesia[2]. The incidence of side effects seems to be low—possibly as little as 1 or 2 per cent. Among the side effects which have been reported are nausea, dermatitis, drowsiness, dizziness, vomiting, and abdominal bloating or cramps.

When Lomotil was made available to us for clinical studies, we used roentgenograms to study its effect on the gastrointestinal transit of barium in human patients. Control studies on a patient with an ileostomy

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*Furnished as *Lomotil* through the courtesy of the G. D. Searle Company, Chicago, Illinois.

Table 1

Age and Sex Distribution

Age (Years)	Sex M	F	Total No. Patients
2 to 9			2
10 to 19			6
20 to 29			11
30 to 39			22
40 to 49			19
50 to 50			12
60 to 69			6
70 to 72			1
Unknown			2
Total	46	35	81

showed that the barium reached the ileostomy opening in one hour (fig. 1a). Following the administration of 20 mg. of Lomotil during a 24-hour period, the barium meal did not reach the ileostomy opening in five hours (fig. 1b). In another patient the control barium meal entered the colon 30 minutes after ingestion (fig. 2a). Following a similar dosage of Lomotil, the barium meal did not reach the colon in four hours (fig. 2b). In neither case was there a demonstrable delay in gastric emptying, but in both cases the transit time through the small intestine was decidedly increased.

Clinical Study

Methods and materials

Table 2

Duration of Therapy

Months	No. Cases
1	26
1	20
2	5
3	3
4	10
5	7
6	7
7	7
8	1

To date, we have used the drug in 81 patients, ranging in age from 2 to 72 years (table 1). All were having active diarrhea, and most of them had a pattern of chronic diarrhea which had failed to respond to a variety of symptomatic measures, including diet and the usual constipating drugs. During the study, each patient was permitted to continue on his previous diet, but received no antidiarrhea, drugs except Lomotil. This was given in doses of 20 to 80 mg. daily for periods of one week to eight months (table 2). The patients kept a daily record of the number and character of their stools and the presence or absence of blood, mucus, pain, cramping, gas, and distention. They also noted any untoward manifestations such as nausea, vomiting, change in appetite, fever, and dermatitis which could be considered as evidence of reaction or sensitivity to the drug.

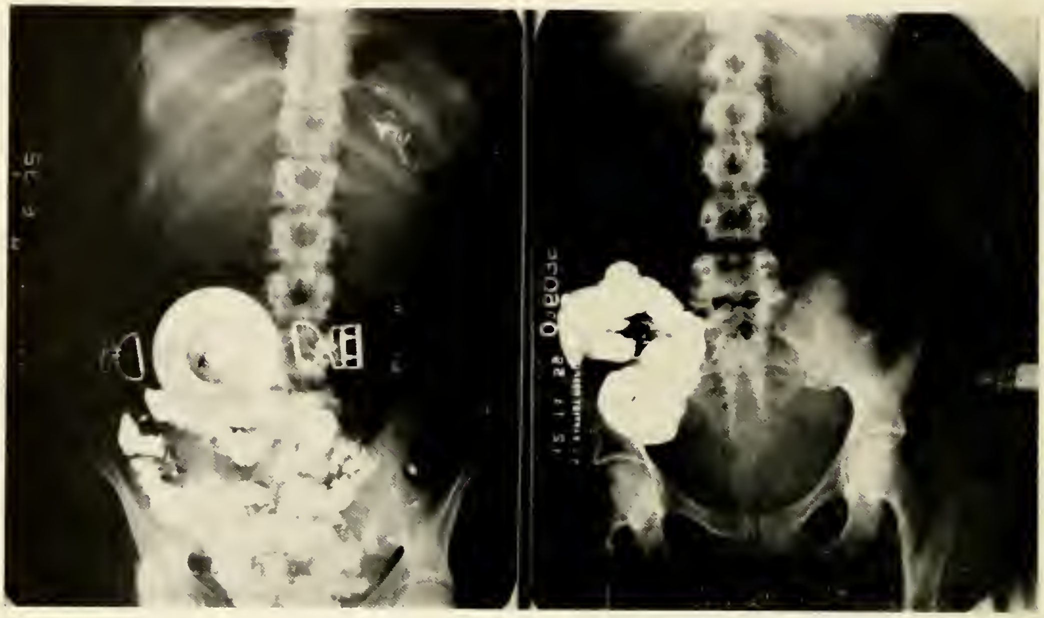
Results (Table 3)

Thirty-eight of the 81 patients (47 per cent) had excellent results, manifested by a prompt decrease to normal in the number of stools or, in some instances, constipation requiring a decrease in the dosage of the drug. Definite improvement was noted in an additional 20 patients. Altogether, 58 of the 81 patients (71.6 per cent) obtained sufficient relief to justify continuation of the medication for prolonged periods. Twenty-three (28.4 per cent) were unimproved. The highest percentage of good results (80 per cent) was obtained in the 30 patients with functional diarrhea. Seventy-five per cent of the 12 patients with regional enteritis had an excellent or fair response. The results in ulcerative colitis were somewhat less impressive than the over-all results, although 63 per cent of these 27 patients could be considered as

Table 3

Results of Treatment

Diagnosis	Excellent	Fair	Unsatisfactory	Total No. Cases
Ulcerative colitis	10	7	10	27
Regional enteritis	6	3	3	12
Functional diarrhea	18	6	6	30
Post-surgery diarrhea	4	1	1	6
Miscellaneous	0	3	3	6
Total	38	20	23	81



1. A. Control roentgenogram taken one hour after ingestion of a barium meal, showing barium at the ileostomy opening. B. Roentgenogram made in the same patient following the administration of 20 mg. of Lomotil during a 24-hour period. Five hours after the ingestion of a barium meal, the barium has not reached the ileostomy opening.

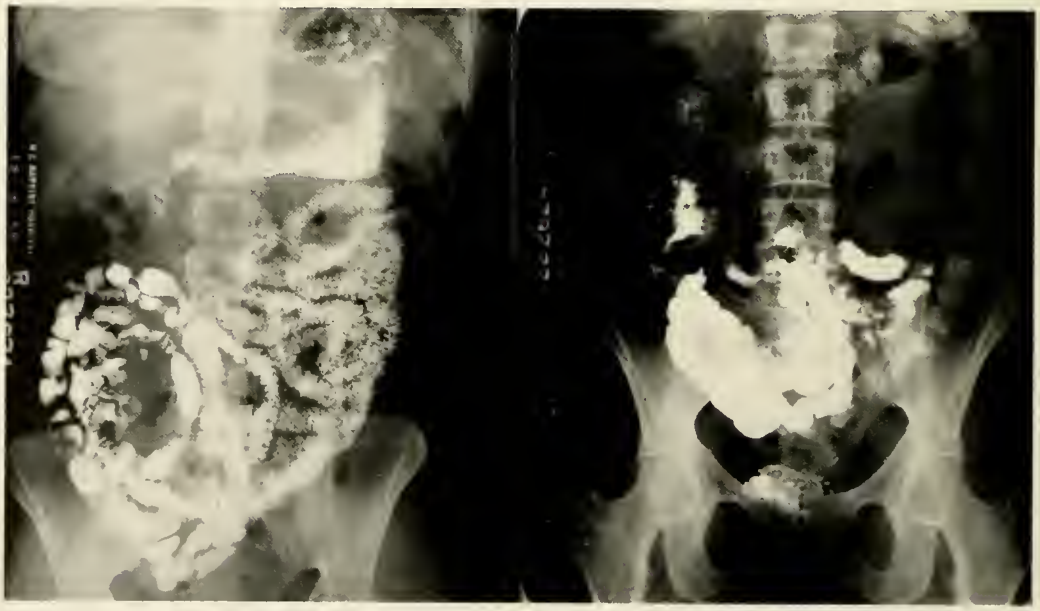


Fig. 2. A. Control roentgenogram taken 30 minutes after the ingestion of a barium meal in a patient with functional diarrhea. The barium column is in the colon. B. Roentgenogram made in the same patient following the administration of 20 mg. of Lomotil during a 24-hour period. Four hours after the ingestion of barium, it has not reached the colon.

having an excellent or fair response. The poorest results were obtained in the 6 patients with miscellaneous disorders, which included ileocolitis, extensive small bowel resections, and chronic pancreatitis with steatorrhea.

The alleviation of symptoms was usually prompt, occurring within 24 to 72 hours even in the long-standing chronic cases. Although the optimal dosage varied, 20 mg. per day proved highly satisfactory in most instances. The patients were usually able to regulate the dosage according to their symptoms. One 9 year old child who received 20 mg. on three consecutive days did not have a stool for 13 days, and then returned to fairly normal bowel habits.

Table 4

Side Effects

	No. Cases	
Nausea	8	
Rash	4	
Itching, coma	1	(patient with cirrhosis)
Total	13	

Side effects (table 4) were relatively infrequent, and when they occurred, were minor in nature and usually disappeared without interruption of treatment. Nausea and dermatitis were the ones most frequently noted. No changes have been observed in weight, blood counts, or urinalysis. One patient with advanced portal cirrhosis had two episodes of coma during the time Lomotil was being administered for concomitant diarrhea.

In all cases, treatment was discontinued by abrupt withdrawal of the drug. No withdrawal manifestations were noted.

Comment

Because no specific therapy for chronic diarrhea has been available, patients have to be treated with symptomatic measures, which are often disappointing.

The constipating properties of Lomotil are clearly demonstrable by roentgen examination. Clinical studies reported by 28 investigators in this country to date have indicated that good relief was obtained by 68.4 per cent of the patients receiving the drug, and some relief by an additional 8.3 per cent. As might be anticipated, therapy with this drug is most successful in patients with diarrhea of functional origin or of relatively short duration.

In our series, an excellent or fair result was obtained in 80 per cent of the patients with functional diarrhea. In the patients with chronic organic disease, a wealth of information concerning the efficacy of various antidiarrheal preparations was available for comparative evaluation, thus obviating the necessity for including a placebo in the present study. A surprisingly satisfactory response was obtained in 75 per cent of the patients with regional enteritis and in 63 per cent of those with ulcerative colitis, all of whom had failed to respond to other measures. In each group, administration of the drug was followed by a significant decrease in the frequency of stools.

The only serious complication of therapy was coma in a patient with cirrhosis. Studies to determine whether the coma was related to drug therapy could not be done at the time. Caution should be used, however, in administering the drug to patients with hepatic insufficiency and impaired liver function.

We noted no tendency to addiction, even in patients receiving up to 40 mg. per day for periods up to eight months. Other investigators have noted that such patients, when challenged by Nalline, exhibit no symptoms of withdrawal[3].

In view of the fact that the material has no taste, more recent preparations have been combined with atropine to prevent deliberate overdosage.* The drug can be administered to children by fractions or multiples of the present 2.5 mg. tablet, or in a liquid preparation. Lomotil is available in 5 mg. and 2.5 mg. tablets, and in a liquid containing 2.0 mg. per cubic centimeter. The dosage can be adjusted to an optimal level as soon as the diarrhea is checked.

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*The dosage form supplied early in this study contained no atropine. In reporting results, however, no distinction is made between patients given pure diphenoxylate with atropine (Lomotil), since no difference attributable to the trace of atropine present was discernible.

Summary and Conclusion

Clinical studies with diphenoxylate hydrochloride (Lomotil) have shown that it has a constipating effect in a significant number of patients with organic and functional disorders characterized by established patterns of diarrhea.

In two patients, roentgen studies of the gastrointestinal transit of a barium meal demonstrated that Lomotil significantly increased the transit time through the small intestine, without producing a demonstrable delay in gastric emptying.

Although the drug is chemically related to meperidine, no analgesic or addicting property has been noted. Patients under therapy for as long as eight months showed no withdrawal symptoms when the medication was abruptly discontinued.

Diphenoxylate hydrochloride (Lomotil) has proved to be a valuable aid in the management of a high percentage of patients with acute or chronic diarrhea due to functional or organic disease.

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Pregnancy in a Case of Adrenal Hyperplasia

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The first breakthrough in the medical treatment of adrenal hyperplasia came in 1950, when Wilkins and associates[1] reported the case of a female pseudo-hermaphrodite with congenital hyperplasia of the adrenal whose urinary 17-ketosteroids were depressed from an average of 48 mg. to 6.5 mg. per day by the daily administration of 100 mg. of cortisone. The authors suggested at that time that cortisone may be of value in treating the adrenogenital syndrome. Subsequent studies by Wilkins[2] proved this assumption to be correct.

In 1956 Yamashita and Kozakae[3] reported the first case of congenital adrenocortical hyperplasia in which the patient, while on cortisone therapy, became pregnant and was delivered at term.

In 1954 Jones and Jones[4] classified cases of adrenal hyperplasia in association with apparently related disorders of diminishing severity and with different times of onset. In the first group were cases of female pseudo-hermaphroditism due to congenital adrenal hyperplasia; in the second, postnatal virilization due to adrenal hyperplasia; in the third, postpubertal hirsutism, oligomenorrhea, and infertilility associated with elevated 17-ketosteroids, possibly due to adrenal hyperplasia; and in the fourth, hirsutis, oligomenorrhea and infertility, but normal 17-ketosteroids. Patients in all four groups responded to cortisone therapy.

At about the same time Greenblatt[5] pointed out that in some women cortisone treatment resulted in a diminution of hair growth.

Bartter[6] first suggested that the primary defect in congenital adrenal hyperplasia lay in the inability of the adrenal cortex to synthesize hydrocortisone readily. This theory has been amplified by other investigators[7] who indicate that the metabolic defect is caused by an enzymatic block in the hydroxylation of progesterone to hydrocortisone. The cause of the block is not clear.

It is theorized that the pituitary attempts to compensate for this defect in the metabolism of hydrocortisone by increasing the production of adrenocorticotrophic hormone, which stimulates the adrenal cortex to increase its output, primarily of androgenic-type steroid. Exogenous hydrocortis-

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one or cortisone inhibits the output of adrenocorticotrophic hormone by the anterior pituitary, whereas the adrenogenic steroids are relatively ineffective in suppressing this action. Thus lies the rationale behind the use of cortisone in the treatment of adrenal hyperplasia.

The case presented here would seem to fall into the third category of the classification suggested by Jones and Jones, since the condition apparently became known in the postpubertal stage, and in the form of hirsutism, oligomenorrhea, and sterility associated with a high level of urinary 17-ketosteroids.

Case Report

The patient, a 21 year old woman, was first seen in July, 1959, with a chief complaint of amenorrhea and sterility. The menarche had taken place at the age of 11, with vaginal bleeding occurring at intervals of one year to three months until the age of 15, at which time cyclic bleeding at about 30-day intervals developed. Menses continued at fairly regular intervals until June, 1958, at which time, following three months of amenorrhea, the patient experienced excessive bleeding. A dilatation and curettage was performed, and the patient was told that she had been pregnant, although there had been no symptoms of pregnancy. This diagnosis was not definitely established. The dilatation and curettage was followed by six months of cyclic bleeding, then by a three-month period of amenorrhea terminating in heavy bleeding. It was during a four-month period of amenorrhea that we saw her first. Of significance was a history of marked increase in hirsutism following the amenorrheac phase 18 months previously.

The patient was a moderately obese woman with acne of the torso, and with black hair, a mustache and sideburns. There was a heavy growth of hair on the abdomen, groin, perineal area, and legs. The weight was 212 pounds.

Pelvic examination

The clitoris was large but not out of proportion to the remainder of the external genitalia. The cervix was nulliparous and epithelized. The uterus was anterior, of normal size, and very hard. Both ovaries were palpable, and were thought to be slightly enlarged.

Examination of the endocervical mucus showed a ferning pattern, indicating estrogenic activity only. A urinary 17-ketosteroid determination was reported as 22.8 mg. per 24 hours. Norlutin, 10 mg. daily, was given for a five-day period, after which vaginal bleeding occurred.

Accessory clinical findings

The patient was admitted to the hospital for further studies. Laboratory data included a negative urinalysis. The hemoglobin was 12.2 Gm. A VDRL test was negative. The white blood cell count was 7,900, with a normal differential. Intravenous pyelographic studies showed a normally functioning urinary tract, without evidence of a suprarenal mass. Skull roentgenograms demonstrated a normal sella turcica. A 24-hour controlled urine specimen was found to contain 40.5 mg. of 17-ketosteroids. Following the intravenous infusion of 40 units of ACTH over a four-hour period, a second urine specimen was obtained and 17-ketosteroids were reported as 42 mg. per 24 hours. The cholesterol was 186 mg. per 100 ml. The basal metabolic rate was minus 8 per cent.

The history, physical examination, and laboratory findings strongly suggested adrenal hyperplasia.

Course of treatment

The patient was given a daily dosage of 12 mg. of Kenacort for two days, reduced to 8 mg. thereafter. On the fifth day of treatment, the urinary 17-ketosteroids measured 9.3 mg. per 24 hours. Approximately one month after the beginning of therapy ovulation occurred as determined by examination of the endocervical mucus. The Kenacort was reduced to 2 mg. daily, and amenorrhea ensued for the next two months. When the dosage was increased to 4 mg. daily, vaginal bleeding ensued. On this dosage the patient had another episode of vaginal bleeding, preceded by premenstrual symptoms. Endocervical mucus again indicated ovulation. The last menstrual period had been March 21. Amenorrhea followed, and in two months the uterus was palpably enlarged. The patient reported signs of fetal life approximately five months after the March episode of bleeding, and at this time the size of the uterus was commensurate with a five months' gestation.

Four to six weeks after the start of steroid therapy, the patient reported a diminution of the hirsutism. This observation was not shared by the examiner. In two months the patient was convinced that the hirsutism was diminishing, and after four months the improvement was quite obvious to the examiner. This improvement continued, and a heavy mustache became a faintly visible fuzz.

The antepartum course was essentially uneventful except for some retention of fluid throughout pregnancy, which responded well to diuretics. Kenacort, 4 mg. daily, was continued. At six months' gestation, the 17-ketosteroids were 13.5 mg. per 24 hours, and at eight months were 12.6 mg. per 24 hours. In view of these high-normal values, the dosage of Kenacort was not reduced.

Labor and delivery

On January 15, 1961, the patient was admitted in labor at what was thought to be at or near term. Contractions were hard, and occurred at four-minute intervals, but the presenting part remained out of the pelvis and the cervix was slow to dilate. There was little retraction of the lower uterine segment. After 20 hours of labor, the membranes ruptured artifically. Cervical dilatation continued slowly. After a period of rest and the administration of fluids, an effort to stimulate labor by maintaining an intravenous Pitocin drip for six hours met with little progress in spite of hard, though ineffectual, contractions. At this point, a diagnosis of primary uterine inertia was made and an elective cesarean section was carried out. During labor the patient received 100 mg. of hydrocortisone intramuscularly every 24 hours. A 7 pound, 2 ounce female infant was delivered by low transverse cervical cesarean section, without difficulty. The infant was examined by a pediatrician, who reported no anatomic nor physiologic abnormalities. The postoperative course was uncomplicated.

Postpartum period

Laboratory data in the postpartum period included a hemoglobin of 12.8 Gm. and a white blood cell count of 14,000, with a normal differential. The carbon dioxide combining power was 6.7 volumes per cent, or 3 mEq. per liter. Chlorides were 100 mEq. per liter, sodium 142 mEq. per liter, and postassium 3.3 mEq. per liter. The 17-ketosteroids one week after delivery were 10.1 mg. per 24 hours. During this time the patient had continued to receive 8 mg. of Kenacort daily.

Summary

The case presented is thought to be a case of acquired postpubertal adrenal hyperplasia treated effectively with corticosteroids, with resulting ovulation, pregnancy and delivery. There was marked lessening of hirsutism and improvement in the acne while the patient was on corticosteroid therapy. Urinary 17-ketosteroids fell from a high of 42 mg. per 100 ml. to 9.3 mg.

It is not clear why the 17-ketosteroid level did not rise after ACTH was given. It is thought that the marked drop after the administration of corticosteroid ruled out adrenal tumor and favored adrenal hyperplasia. The patient received 4 mg. of Kenacort daily throughout pregnancy. This dosage was not reduced, since the 17-ketosteroids remained at high normal levels on this amount. The recurrence of acne during pregnancy would be in keeping with the increase in 17-ketosteroids over the pre-pregnancy levels on the same dosage of Kenacort.

The patient was delivered by cesarean section because of primary uterine inertia as an obstetric complication and not because of the metabolic disturbance. The infant showed no untoward effects from the relatively high androgenic levels during pregnancy or from the corticosteroid therapy given the mother.

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The Development of Malignant Hypertension Following Nephrectomy

Complete Relief by Reconstructive Vascular Surgery on the Remaining Ischemic Kidney

A Case Report

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The purpose of this paper is to report a case—as far as we know, the first in literature—in which malignant hypertension developed following the removal of a pyelonephritic kidney, and was completely relieved after normal arterial blood flow to the contralateral ischemic organ was restored.

Case Report

A 36 year old housewife and machine operator was admitted to Charlotte Memorial Hospital on March 22, 1961, with severe abdominal pain. She had had acute pyelonephritis during pregnancy 15 years previously. Five years prior to admission she began to have pyuria, and was treated with various antibiotics. Thirty-six hours before admission severe right-sided abdominal pain with nausea, vomiting, and pyuria developed. Examination showed pyelonephritis with multiple stones in a hydronephrotic right kidney and spontaneous perforation and extravasation of urine. The function of the left kidney appeared to be normal. A right nephrectomy was performed and was tolerated well by the patient. The pathologic report of the kidney was as follows: "Renal lithiasis, dilatation of the calyces, chronic pyelonephritis, focal subcapsular hemorrhages and necrosis." The blood pressure on the day of admission was 130/80; on the day of discharge it was 160/88.

For six months afterwards the patient felt reasonably well. Then she began to have generalized headaches which were associated with nausea but with no vomiting at first. In December, 1961, she was found to have a blood pressure of 180/115. She was given reserpine and Hydro-Diuril, but the blood pressure rose to 220/130. In January, 1961, she had to stop work, her headaches having become more severe. During the month of February she had been incapacitated by generalized, prostrating headaches associated with nausea and vomiting. There was no fever, and urine examination showed no pyuria.

She was readmitted to the hospital on March 22, 1961. Physical examination at this time showed a thin pallid, chronically ill, 36 year old white woman who was complaining bitterly of severe headache. The temperature was 37° C., the pulse rate 100, and the blood pressure 220/130. She

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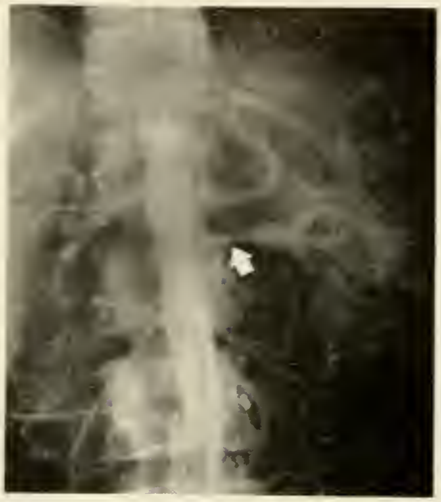

Fig. 1. Retrograde abdominal aortogram. The arrow marks the stricture of the solitary left kidney artery.

had a healed nephrectomy scar on the right. Routine physical examination otherwise was noncontributory.

Ophthalmoscopic examination disclosed multiple retinal hemorrhages and exudates bilaterally. Examination of the urine disclosed a specific gravity of 1.015, 1 to 3 red blood cells, and 10 to 15 white blood cells per visual field, and no albumin, sugar or ketones. The blood sugar was 100 mg. per 100 ml. and the blood urea nitrogen 26 mg. per 100 ml. The hemoglobin was 14.2 Gm., the hematocrit 45 per cent, and the white blood cell count 10,000, with 44 segmented cells, 47 lymphocytes, 6 eosinophils, and 3 monocytes. A PSP test showed 55 per cent excretion of the dye in one hour. The serum calcium was 9.6 mg. per 100 ml.

An intravenous urogram indicated satisfactory function of the left kidney. Roentgenogram of the chest and upper gastrointestinal tract showed no abnormality. An electrocardiogram revealed prolongation of the QT interval and left ventricular enlargement.

The patient's history and physical findings suggested that the hypertension might be the consequence of a constriction of the artery to the remaining left kidney. The aortogram showed filling of the abdominal aorta and all its main branches except the renal arteries. Retrograde aortic catheterization was performed on the following day. A no. 8 Lehman catheter was introduced through the right femoral artery up to the level of the left kidney; 20 cc. of a 90 per cent solution of Hypaque was injected through the catheter, and the flow of dye was followed by four-frames-per-second high speed roentgenograms. The abdominal aorta and its branches were again well outlined. The renal artery showed a definite constriction of the lumen just distal to its origin from the abdominal aorta. The length of the stricture was 6 mm.

To relieve this constriction, an operation was performed on May 11, 1961. Under endotracheal anesthesia the patient was placed on the table in the supine position. She was packed in ice and the body temperature was lowered to 28°C. A median laparotomy was performed. The abdominal aorta with its branches was dissected free. The left kidney was exposed. The origin of the left renal artery was found to be firm, nodular and narrow, and no pulsation was felt over it. The artery was cross-clamped, and 20 cc. of saline solution containing 20 mg. of Heparin was injected distal to the occluding clamp. The abdominal aorta was also cross-clamped distal to the renal artery, and a Dacron vascular graft was anastomosed end to side of the abdominal aorta and to the renal artery distal to the obstruction. After the aorta was released from the clamp, good pulsation of the renal artery was observed. The parietal peritoneum and the abdominal wall were closed in a routine manner. The patient tolerated the procedure well and was moved to the recovery room, where her body temperature was gradually increased to 36°C.

Recovery was uneventful. Her blood pressure returned to normal, and six weeks following her discharge from the hospital ophthalmoscopy showed complete disappearance of the changes previously described.

Discussion

The cause of renal artery stenosis in this case could not be determined with certainty.

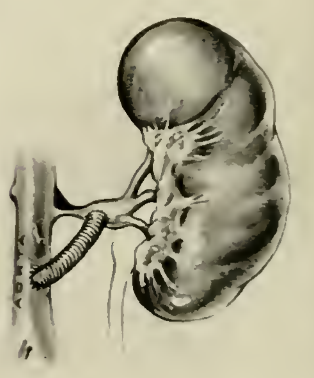

Fig. 2. Schematic presentation of the operative procedure. The crimped vascular graft connects the orta to the distal renal artery.

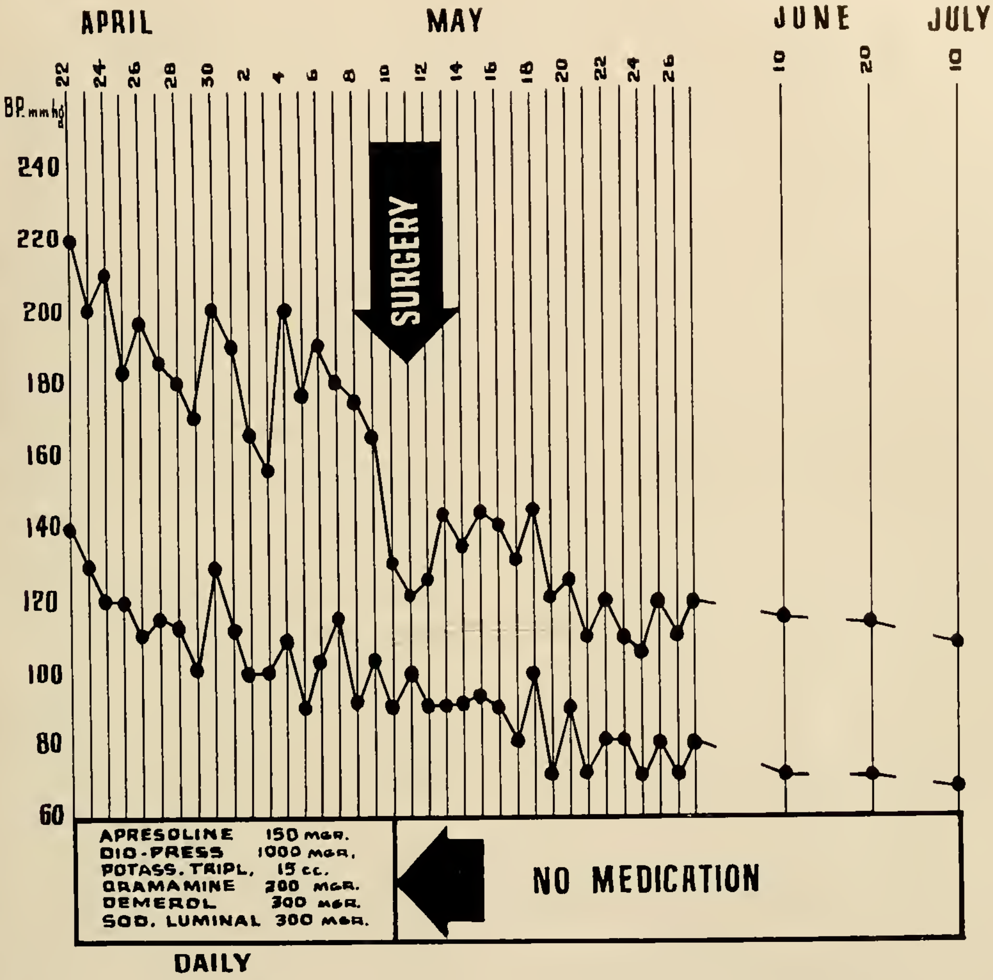

Fig. 3. Changes in the arterial blood pressure before and following surgery.

The absence of arterial hypertension before the right nephrectomy indicates that it might have been a consequence of the operative trauma itself. Development of constrictive scarring round the renal artery following surgical intervention[1] or common trauma[2] has been described by several authors. The possibility that the stricture of the left renal artery was already present, the Goldblatt-syndrome being triggered by the removal of the right kidney, is also to be considered. The "protective role" of the contralateral organ was well documented in the experiments of Kolff[3].

This case also demonstrates that if unilateral renal vascular disease is suspected on the basis of the history and physical findings, its presence cannot always be excluded by a normal intraevnous urogram or by other tests of renal excretory function. Separated renal function studies with comparison of water and sodium excretion of each kidney may strengthen a clinical impression of unilateral disease of the main artery, but are not diagnostic. Excretion patterns of patients with bilateral stenosis of the renal arteries may be indentical to those with unilateral disease of the main renal artery. Obviously, such studies were not possible in this patient[4].

The only proof of the presence or absence of constriction of the renal artery is adequate angiography. The reliability of this diagnostic method exceeds that of operative exploration. We believe that the dangerous of this examination are somewhat overemphasized in the literature. If it is done with the necessary experience and maximum caution, untoward effects are rare and the value of the information gained far exceeds the risk involved. Contrast-filling of the renal arteries may be done by two different methods: percutaneous needle-puncture of the lumbar aorta, or by insertion of a catheter retrogradely through the femoral artery. There have been no severe complications or deaths with either technique in our institution, nor in the series of 500 patients of Poutasse[5].

The following indications for renal angiography are generally accepted:

1. Disparity in the size of the kidneys or lag in appearance of excretory function on one side.
2. Hypertension in a patient less than 35 years of age, or malignant hypertension developing suddenly in a patient more than 55 years of age.
3. Nonfamilial hypertension of recent onset in any patient, regardless of age, with rapid progression into the malignant phase.
4. Hypertension developing after an episode of flank or abdominal pain.
5. Hypertension developing after trauma or abdominal surgery, especially urologic operations.

If the angiogram shows impairment of the renal blood flow, an operative plan should be established which offers maximum benefit for the patient, with the simplest technique. All efforts should be made to preserve renal tissue and function. Despite the fact that in some cases nephrectomy is inevitable, renal conservation through direct revascularization is the treatment of choice. Certainly, if the process is bilateral[16], or if there is a solitary kidney as in the case presented, this is the only effective method available.

Summary

Malignant hypertension developed in a previously normotensive 36 years old woman several months after nephrectomy for pyelonephritis and nephrolithiasis. Retrograde aortic catheterization permitted radiographic demonstration of a stricture of the renal artery supplying the remaining kidney. Following reconstructive vascular surgery, the patient's blood pressure became normal again.

The physiologic features, diagnosis, and surgical importance of hypertension of renal vascular origin are briefly discussed.

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Folk Medicine in an Isolated Modern Community

JAMES EARL SOMERS, M.D.

CHAPEL HILL

This paper is a report on folk medicine and its contact with contemporary medicine as observed in a small community on the Cherokee reservation in the mountains of Western North Carolina. The work was done as part of a three-year field study of the culture of the eastern band of Cherokee Indians, under the directions of Dr. John Gulick, Department of Anthropology, University of North Carolina, and financed by a grant from the Ford Foundation. My work was done during a short period in the summer of 1959, the second year of the project.

The community of Big Cove is about 9 miles from the town of Cherokee, at the end of a dusty, gravel road, mostly up grade. Several miles of National Park further isolates this part of the reservation, known for its full-blooded Indians.

This community was picked for study as probably representing the least acculturated of the Indian groups. There is a grammar school in the lower part of the cove, where for a number of years a public health nurse has held clinics, visiting in the homes whenever possible. The government center hospital is in the town of Cherokee. It is a modern, 25-bed structure, with excellent equipment and two full-time staff physicians. Some of the nurses are part Indian. Free medical facilities are available only to indigent Indians, but this provision excludes only a few residents of Big Cove.

General Attitudes

Some of the general cultural problems and attitudes in the less acculturated inhabitants as reported by the doctors and nurses and observed by me were as follows:

There was little concern about seeking or continuing medical treatment except for the relief of severe symptoms such as acute discomfort or high fever. This trait makes the practice of preventive medicine difficult, and is a reflection of the culture, in which small provision is made beyond present needs. There is no follow-up after an illness or a birth, so that each visit is a new one rather than a continuation of the doctor-patient relationship. Patients refuse to follow recommendations if they disapprove of them. They expect immediate results, or at least a prediction of what to expect, and when.

The reality judgment is faulty—for example, where the choice is between medicine and soda pop and there is not enough money for both, the child's preference rules.

A recent general cultural problem is faith healing, as practiced by some of the fundamentalist cults. An ethnologist reports on a healing service at which a small child and a baby with colds were brought forward, anointed with commercial olive oil, and prayed over long and loud. The preacher, a white woman, said that she had had female trouble for years, and was not cured by the doctors, who gave her only some pills. She said that healing took place by faith in God.

Another problem is what might be called family medical parctice. When a child objects to entering the hospital, the mother often insists on being admitted, too. I was present on one occasion when a child of 4 was admitted with congenital heart defect and pneumonia. The mother refused to leave him; so she too was admitted, along with an 18 month old sister, who was still nursing.

One of the cultural criteria of good mothering is a fat baby. For this reason, babies are fed fattening foods from the table as soon as they will eat them, and this often is less than one month. Gravies, soggy grease and starch concoctions are commonly fed. Some mothers chew the food and then give it to their infants at the age of one month. One mother reported that her baby

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From the Department of Psychiatry, University of North Carolina School of Medicine, Chapel Hill.

had not been fat. An aunt had predicted that the infant would not live long, but it was the healthiest child she had had. Another mother, who had married into the Big Cove community from another part of the reservation, reported having been concerned when her mother-in-law fed her first child from the table when she was two weeks old. Since the child appeared fat and unharmed, the mother had continued this practice with her other seven children.

Plumpness is similarly admired in married women as a sign of prosperity. This is a problem in any medical treatment of which diet is a part, particularly diabetes, which is a common disease and almost impossible to control. I might add that the control of diabetes is even more difficult in men, because of the intake of alcohol.

The problem of communication is uncommonly difficult in Big Cove. The public health nurse reports that doctors are often criticized for not treating symptoms they did not know were present. This happens because of the belief that doctors should know everything without being told. A mother had complained that nothing was done for her hemorrhoids during her last hospitalization. On being questioned, she admitted that she had not reported the condition, feeling that since she was in the hospital the doctor should know and treat everything.

Another medically significant trait is the culturally determined reactions to pain and illness. The restraint of strong feelings, even in severe pain, was evident, at least as far as outsiders are concerned. Informants felt that the more culturally Indian the patient was, the less feeling or reaction to pain he would show. This became obvious to me while I was attending three Indian women in active labor. I was timing the contractions for frequency and heard only muffled sounds with the strongest contractions. My observation was confirmed by the public health nurse. In her long experience in obstetrics, this was the only labor room she had ever encountered that was quieter than the rest of the hospital.

The routine sedative available in labor is Demerol and a Trilene mask. The nurses reported giving Demoerol routinely, since it was seldom requested. The doctor reported that it was difficult to gauge the progress of labor without the increasing activity of of the mother. This reluctance to express pain was common in other medical situations, such as fractures, and he gave anesthesia on the basis of past experience rather than the patient's reaction to pain. The doctor told me of a patient he had referred to a specialist in Asheville. A complicated fracture was set without anesthesia, as the patient made no complaint during the whole procedure.

The control of emotion was striking also in situations such as coming home from the hospital, as with a new baby. I accompanied one of the Indian matriarchs home after she had been hospitalized for a week. We entered the cabin, spoke to the adults present, and began talking much as if there had been only a brief interruption in conversation. No feeling about coming home or concern about the hospitalization was expressed.

The Medicine Man

In Big Cove, the role of the medicine man has suffered considerable acculturation. The medical center, the efforts of the public health nurse, and the influence of the school have undermined his position, but the practice of conjuring and treating persists.

There were three men who function occasionally in the role. The most active is ——————, who has in his possession the ceremonial book written in Sequoyah's Cherokee alphabet. The ceremony is the ritual that, according to the degree of illness, accompanies the medication. He reported that he got the ceremonies from an old man who gave him the written directions and nothing more. Others say that you have to be told and then practice the ritual. The medicine man needs faith to be successful in curing, but faith on the part of the patient is not necessary. The prestige of the medicine man has suffered considerably with the more acculturated Indians, who openly ridicule his activities. His feats, however, are sometimes widely discussed, and at times he leaves the reservation to perform "cures."

During the first summer of this project, an anthropologist sought and received the services of one of the medicine men. I extracted the following account from his field notes:

> About 5 p.m. I picked up the C————s and took the road up the Cove. On the way I described the symptoms to Mrs. C————, who in turn translated them to her husband. Symptoms were a heavy chest cold and a wrenched back which I had suffered that morning in helping lift a log onto a truck. We arranged for me to return the next evening for medicine. I gathered that I was to wait to receive part of my remedy, and apparently C—— went up in the mountains to secure the necessary roots and bark.
>
> After 20 minutes he came down the hill with a very long birch pole which he deposited on the wood pile, and after another five minutes he brought the medicine which was to relieve the sore back. The bundles consisted of splints of bark about 7 inches long and 1½ inches wide, representing three species of trees. Later I discovered in the interior of the bundle one small root, perhaps "yellow root," tied in a curious double-eight knot. This was the only item that was not in pairs or multiples.
>
> The directions were to boil this in about two cups of water for ten minutes, drink a cup of it before going to bed and on waking during the night. I asked Mrs. C———— how much I owed C———— for the medicine, and she replied, "Later." She indicated that payment would depend on the effectiveness of the cure. Walker told his wife, who translated to me, that I was to return the next evening for a potion to cure the cold. He disappeared into the house briefly and reappeared with what seemed to be three small ginseng roots, indicating that they came from the top of the mountain and that this potion would have to be prepared at the C———— house. I agreed to return at 7:00 the next evening for this part of the cure.
>
> Later that evening I boiled the concoction as directed and drank half a cup of liquid that remained on the bark. The effect was masked perhaps by the fact that I found it desirable, as soon as possible, to dilute it with three additional cups of water. Whether it was the added water or the potion which produced the diuretic effect within the next hour unfortunately cannot be known. I was honestly able to report a slight improvement in the backache next morning.
>
> Later I asked an informant the correct procedure for paying Indian doctors. She stated that no money could change hands or the cure would be spoiled; it was correct to give a cloth on the first visit, and it should be white if it was for medicine. If the cure was successful, it would be appropriate to give something to eat, possibly a little pork.
>
> When I returned to see C——— I told him my back felt better. He tried to explain, half in English and half in Cherokee, how the medicine worked. He inscribed with his finger a small circle in the median saggital line in the vicinity of the solar plexus, and ran his finger around the region of the kidney. At this point he went into the house and was heard pounding something for about 10 minutes. Finally he returned with a small brown package which he handed to me and directed me to pour about a quart of hot water over the roots contained therein, and to drink this potion before I went to bed and during the night. I gave him the white handerchief, which he accepted with no visible feelings. When asked if I should return for more medicine, he said after some thought, "Wednesday."
>
> In discussing my treatment later, I was told that in native curing, when the symptoms leave dramatically, a recurrence can be expected; but if they leave gradually, the cure is permanent. Ten days later Walker left the medicine at the Field Station for me. There were two packages wrapped in white paper, one containing medicine for the back (strips of bark) and the second pounded root, probably similar to that prescribed for my cold except that it was much fresher, having been recently dug. It lacked the musty odor and yellow color of the former.

The ailing anthropologist recovered and paid his doctor with a small pork roast.

Medical Lore

There is a general body of medical folklore, parts of which I have compiled from all of our field notes. Where possible, I cross-checked the information, and at a later visit I checked much of it with Lloyd Sequoyah, the medicine man generally thought to be the most consistent and the most honest. I also obtained information on some specific cures.

Procreation

A great deal of the folklore seems to center in procreation and children. One of the food taboos reported was that women should not eat pheasant from puberty until after the menopause, as this bird was known to be most prolific, and women who ate it would have too many children. There were two food taboos for women who are menstruating: one, fish (no explanation obtained), the other, game that had been shot with a gun rather than with bow and arrow. Game shot with a gun bleeds more and allegedly causes excessive bleeding during menses.

There was an unknown herb to prevent pregnancy, but apparently there was a great deal of strong feeling about using it. Two informants reported that they had been unable to learn anything about it because "it was against God's laws, and you should not interfere in this way." Another said that mothers and mothers-in-law were forbidden to tell their daughters about it, but that it was all right to tell someone who was not of kin.

To help a woman become pregnant, a potion prepared from the ball that grows on the stalk of a certain weed is used. Inside the ball is a worm which represents the fetus; the ball, the belly. In some situations, the husband cuts a little of the worm and puts it into his wife's drink without her knowledge. She drinks this potion and becomes pregnant with the next intercourse. Once is enough: it need not be repeated after the first child.

Two methods of telling the sex of an expected child were reported. If the youngest sibling's first word referred to its mother, the child would be a boy, and vice versa. Another method was to observe whether chiefly boys or girls played around the pregnant woman. My informant could not remember whether a child of the same or the opposite sex could be expected, but she guessed the latter.

To insure an easy pregnancy, regular medication should be started after the third month. This consists of a brew made from the first leaves that fall from the Buckeye tree, and from bass wood bark. The directions are to boil a bunch of these in half a gallon of water and drink some every morning. Next, go to the creek and get rid of the phlegm (mucus in the throat) until time for delivery. When the moon changes, have the medicine man dip handfuls of water from the creek and throw it in the direction of the flow. This will insure a rapid delivery since, according to an old story, water is the most powerful of forces; it can even move rocks. To further insure rapid delivery, make sure that everyone, at about the time of delivery, walks through doors; however, they should avoid leaning out of windows. Leaning out of a window is similar to the baby coming partially out and going no further. If delivery is hard, make a mixture of seven pumpkin seeds and seven sassafras tips in water, heat, and drink. For swelling during the last months of pregnancy, put certain sticks covered with bark in warm water, but do not boil. Let this sit for a while, then drink in the middle of the night until the swelling goes away.

Two specific complaints that I heard frequently at the hospital involved the routine delivery of babies. One was that no cold liquids should be given the mother during or after delivery. The hospital specifically violated this rule by making ice water available. Folk medicine prescribes that warm liquids, usually prepared by boiling a specific bark, should be drunk during this period. One informant confessed that on her last home delivery she had twice drunk some cold water when her mother was out of the house, and each time had later had violent pains in her stomach.

Another rule concerned the disposal of the placenta or afterbirth. The culture prescribes that it should be buried behind the house in which the delivery takes place. When the remainder of the cord comes off, it should be buried nearby. The hospital, however, disposes of the placenta in the incinerator. Each of the mothers whom I questioned seemed concerned about this, but none knew if anybody had complained to the hospital authorities. The same was true of the cold water.

Infant care

Next to problems with the baby: For crying, dip the baby's toes in water seven times and have the baby drink water. This should result in his sleeping through the night. Another remedy for the same complaint is to put seven hickory balls in a glass of water and have the baby drink it. A third method is to tie a wet dish cloth around the child's neck. This will clear his mind of trouble and result in sound sleep. The latter remedy is also recommended for nightmares in older children and adults.

As an aid in teething, cook squirrel brains and rub them on the baby's gum. This will make him teethe easily and have strong teeth. To help in toilet training (which incidentally is not recognized as a problem), get mite worms about an inch long from rotting hickory trees. Rub these on the child's stomach from one month of age on and he will soon train himself not to wet the bed. For vomiting, have the child drink a warm brew made from a certain bark plant. As a general remedy—what we would call a tonic—the herb, boneset, is recommended.

Specific Remedies

The following are remedies for specific conditions:

"Sickness of the mind" is reported as the most difficult illness to treat. Etiologically, unrest starts in the heart and moves toward the brain. A ghost or outside spirit may cause this to happen and make the person's spirit go wild. He then hears voices and sees things that are not there. Sometimes he believes he is being attacked, and runs away. This is the nearest we came to a description of delirium tremens, despite the evident amount of chronic alcoholism. Persons thus affected are also described as attacking persons ordinarily close and dear to them. Treatment consists of a ceremony to quiet the patient. The ritual involves washing the person's face in water (the most powerful force), which carries away the offending material from the mind.

A second condition, appendicitis, was described as similar to being struck by lightning, and the most effective remedy was a brew of the bark of a tree that had been struck but had grown back together again.

For measles (they were having an epidemic at the time), there were two remedies —both for making the illness light and for causing the rash to break out early, with consequent relief of pain and fever. First, get the grass with the blue flower from under an apple tree, and the heart-shaped leaves of a vine from the same location. Mix these with mint that grows along the edge of a creek, and boil the mixture in a half-gallon of water. Drink half a cup of the potion and repeat at regular intervals until the rash breaks out and the fever goes away. Another brew is made from the bark of the spice tree.

As for the etiology of measles and disease in general, the old folks believed that the disease was carried by the wind, and during an epidemic one should stay inside.

For influenza there were also two remedies. The first was a brew made from wild balsam and wild cherry, and if possible from wild plum. (A mattress filled with the shavings of beaver wood is also helpful.) The second remedy is made from two species of mint, the branch mint and the land mint, and is taken with catnip and boneset weed. Together or alone, they cause sweating, which knocks the fever out.

For heart trouble, or heart cramp, the roots of the snake plant and devil vine are prescribed. As in the case of any severe illness, a ceremony is also needed, but details were lacking. Some ceremonies consist of washing with water, some of scratching the skin with bones or sticks, and some of rubbing, accompanied by certain incantations in the Cherokee language.

For pneumonia, a tea is made from sassafras and spice wood root. For the phlegm which accompanies it, a brew is made from the seven-bark plant and a vine that grows on the bank of a creek.

For a broken bone, a special ceremony is necessary, together with the application of a mixture of sage and broken corn stalk. The directions are to mix the ingredients in a cup of water for 30 minutes. Next make four scratches over the broken bone, suck with the mouth and spit, and then apply the preparation. This makes the bone thread and grow together.

For snake bite apply tobacco juice within one minute. For a poultice to prevent swelling, take bass wood root, beat it with water, apply, and allow to stay for 24 hours. The poultice draws out the poison as it dries.

Two informants told us that the latter remedy has been in disrepute since the medicine man was bitten by a rattle snake the previous summer. Because of the severe pain and swelling, he went to the hospital for treatment. He denied, however, that the treatment at the hospital had been effective, insisting his own treatment effected the cure.

Report from The Duke University Poison Control Center

Jay M. Arena, M.D., *Director*

Vitamin A

Vitamin A poisoning, or hypervitaminosis, is due to the excessive and indiscriminate use of vitamin A concentrates for daily vitamin requirements, acne, and other skin disorders. The toxic factor in vitamin A poisoning is probably the permanently elevated plasma vitamin A level. Clinical hypervitaminosis A develops only after the liver is no longer able to remove from the circulation the excess amount ingested. Hepatic dysfunction may be the basis for the vitamin A poisoning and faulty or limited excretion may contribute to maintenance of high blood levels.

In rats excess vitamin A has been shown to cause acceleration of periosteal proliferation, rapid consumption of epiphyseal cartilage, and remodeling of bone attended by osteoclasis. Hemorrhages due to hypoprothrombinemia are common in vitamin A poisoning in animals, but are rare in humans.

The chief symptoms are a long latent period, irritability, anorexia, scaly skin eruption, pain along the bones most exposed to trauma, swollen legs and forearms, pruritus, alopecia, cracked and bleeding lips, and tender swellings of the skull. Hepatosplenomegaly, hyperpigmentation, persistent severe headache, exophthalmos, papilledema, and a craving for butter may be present. A massive dose of vitamin A may cause an acute, benign hydrocephalus. Roentgenograms have shown hyperostoses of the bones involved, occasional mottling of epiphyses, thinning of the calvarium, decalcification of the skull and vertebrae, and calcification of pericapsular structures.

Treatment consists merely in withdrawal of the vitamin. Most signs and symptoms disappear within a few weeks, but the hyperostoses remain evident for several months after clinical recovery has occurred.

Carotenes of plant origin are readily converted into vitamin A by the liver, where it is stored and from which it is released as needed by the tissues. In the prolonged ingestion of carotene foods, (carrots, yellow squash, sweet potatoes, etc.) and in diabetes mellitus, hypothyroidism, and disorders of the liver, the conversion of carotene may be disturbed, and it may appear in unusual amounts in the blood producing *Carotenemia*, a condition in which the skin (particularly the soles and the palms) shows a yellow discoloration. The sclerae, however, are not involved. Lycopenemia, a condition similar to carotenemia except that the skin is more highly tinted, a deep orange-yellow, is due to high serum and hepatic levels of lycopene from excessive ingestion of tomato juice.

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STATE MEDICAL JOURNAL CONFERENCE

The fifth biennial State Medical Journal Conference was held in Chicago at the Sheraton Hotel, October 30-31. Both the editor and Mr. J. T. Barnes, business manager of the NORTH CAROLINA MEDICAL JOURNAL, attended, and found it well worth while. While little of the conference would be of particular interest to our readers, it is hoped that a few comments will be in order.

The present editor of the *Journal of the American Medical Association*, Dr. John Talbott, and two of the past editors—Morris Fishbein and Austin Smith— were on the program. Dr. Talbott's subject was "What Constitutes a Good Editorial," and Dr. Fishbein spoke on "Medical Writing in State Medical Journals." Dr. Smith, who is now president of the Pharmaceutical Manufacturers Association, discussed state medical journals as advertising media for pharmaceutical companies.

Mr. O. M. Forkert again offered many constructive suggestions about format. Mr. A. J. Jackson, president of the State Medical Journal Advertising Bureau, took a rather optimistic view of the future financial prospects of state medical journals. While recognizing that all these journals have had a decrease in advertising, he thought that brighter days were ahead. Mr. Ted Wiprud, however, thought that the 1959 peak was really an inflationary condition, and that we are now back on a more realistic level, which will probably be maintained for some time to come. Incidentally, Mr. Wiprud deserves credit for arranging the program. He was given an enthusiastic vote of thanks for a job well done.

It is always interseting to note the contrast in the various state journals. Some change their covers every month, some at less frequent intervals; while some, including the NORTH CAROLINA MEDICAL JOURNAL, use the same cover design month after month and year after year. It was consoling to the more conservative members of the Conference to hear veteran editor Morris Fishbein say that content is more important than format. As evidence, he cited the *New England Journal of Medicine*, which is the outstanding state medical journal, but which has the most outdated format.

* * *

THE BOND ISSUE DEFEATED

On November 7 the citizens of North Carolina voted almost 2 to 1 against the 60 plus million dollar bond issue. While this journal does not intend to enter into a political discussion, the subject is of so much general interest that it seems in order to offer a few postmortem comments.

There are a number of possible explanations for the unexpectedly large opposition:

1. So many projects were included in the proposed issue that many said, "A pox on all your houses," and voted against them all.

2. Not enough time and thought had been given to telling the public of the needs of the various projects voted on.

3. Most often heard is that the tax on food

passed by the last legislature aroused so much resentment that many voters took advantage of the opportunity to express their opposition to it.

4. Many think that too much is spent on extracurricular frills in our schools, especially on athletics, and that our educational institutions have not done as well as they should have in really educating our students. In his comments on the torch race (*Winston-Salem Journal and Sentinel* for November 5), Roy Thompson observed that one sentence in a letter written by the student who was chairman of the marathon indicated that there was need for improvement in our educational system. The student said that the runners would have a police escort on their way to Raleigh—but spelled escort "escourt" and Raleigh "Raliegh."

5. The most probable reason of all is simply that people are fed up with paying taxes. The double take of 1960 was followed by the first billion-dollar budget in the state's history. People knew full well that the bonds voted on would eventually have to be paid by taxes, and that still higher taxes would be required. Perhaps many who voted against the bond issue recalled with misgiving Harry Hopkin's dictum, "We will tax and tax, and spend and spend, and elect and elect." At least our state administration has been warned that our citizens want a moratorium on increasing tax rates.

* * *

FISHBEIN COMMENTS ON THE KEFAUVER DRUG CONTROL BILL

In *Medical World News* for October 13 Dr. Morris Fishbein devotes his leading editorial to the Kefauver-Celler Bill. Because he is so well qualified to discuss this bill and because of the acute interest in the proposed legislation, his editorial is quoted in part:

"On Sept. 13, the Secretary of Health, Education and Welfare, Abraham Ribicoff, gave a statement to the Senate Subcommittee on Anti-trust and Monopoly on the Kefauver-Celler bill to regulate the drug industry.

". . . Sec. Ribicoff . . . decries the fact that many pharmaceutical manufacturers imitate successful products introduced by other manufacturers. And in the next breath he condemns monopoly of any product by a manufacturer. He condemns—and I fully agree with him—extravagant advertising and untruthful promotional campaigns. The question is why have not existing laws been sufficient to prevent these abuses.

"I believe the difficulty has been not with the laws but with the utterly inadequate funds allowed to the enforcement agencies for the enforcement of those laws. However, any new legislation that would hinder the pharmaceutical industry from spending what it now spend on research would inhibit medical progress. And legislation that would consider the drug industry as unique from any other industry in its patent privileges inevitably damages American pharmaceutical progress.

"Sec. Ribicoff had much to say about two other matters: the naming of drugs and a need for proof of effectiveness before a product is granted a license by the Food and Drug Administration . . .

"In my years of association with the Council on Pharmacy and Chemistry of the American Medical Association . . . I can remember innumerable instances in which the 17 recognized authorities, including every phase of chemistry, pharmacology, biochemistry, and medical practice found it exceedingly difficult to establish the effectiveness of a remedy.

"A remedy effective in one does for one person may be poisonous in the same dose for another. People are not capable of standardization: the response of the living body is a higly variable factor.

"There are other points which need to be carefully considered. For instance, Sec. Ribicoff feels a new drug should not be allowed on the market until HEW has determined that it will be safe and effective in the diseases and under the conditions of use for which it is offered. One can anticipate the months, or even years, of delay that might ensue because a timid Secretary hesitates to risk his political future in releasing a drug about which there might be a shadow of a doubt. Had such a law been in effect in the past, it is possible that many millions of persons would have died unnecessarily of the conditions controlled by insulin, penicillin, the sulfonamides, diphtheria antitoxin, and many, many other drugs widely used today.

"On certain other matters, however, one cannot possibly disagree with Sec. Ribicoff. There may be instances of abuse in labeling, in exploitation and in promotion of drugs. There may be evasion of existing laws in this as in other fields. However, the sweep of indictment is far too broad. Perhaps the ultimate legislation will constitute something more resembling a rifle aimed at a target instead of a blunderbuss that scatters shot far and wide without being quite sure as to what is intended to be destroyed."

* * *

THE PROBLEM OF RELIEF

When the Social Security program was started in 1936, President Roosevelt said that it marked the beginning of the end of public relief. Like so many other political prophecies, however, it has not come true.

An article in the *United States News and World Report* for September 11 pointed out that the relief payments have now reached five billion dollars a year, and are steadily increasing. In addition, Social Security payments in 1960 were 13 billion dollars, and unemployment pay 2.7 billion.

The relief program has put a premium on illegitimacy and on what government gobblydegook might call a non-desire to seek profitable employment—or, in one word, laziness. Cases of the abuse of relief funds from all over the nation were cited by the *U. S. News and World Report* for September 11. Of particular interest to North Carolinians is a half-page devoted to one county in our state, which spends almost as much for welfare as for schools. The total welfare budget, including state and federal aid, is more than one hundred times as much as the county spent during the depression in 1936 ($599,000 a year, compared with $5,000). Among the examples of the abuse of relief was that of a mother who was getting public assistance for her children, and who hired a Negro maid to look after them while she herself took a full-time job. The captain of a fishing boat who earned from $15,000 to $25,000 during the fishing season drew unemployment compensation for the rest of the year.

Numerous other examples were given, but need not be cited. It was encouraging to note that the article concluded by saying that protests against the rising cost of relief have been pouring into Washington at an increasing rate, and that the Secretary of Health, Education, and Welfare is recognizing the problem. Let us hope that a solution will be found.

* * *

FOOD AND FALL-OUT

In a recent "Strictly Personal" column, Sydney J. Harris introduced a disturbing new thought about bomb shelters: What will happen to our food supply after an all-out nuclear attack? So far, has anyone stopped to think that those who survive the immediate effect of such an attack by using a bomb shelter would be doomed to a slow death by starvation later on? The animals used for food would not be fit to eat, and milk from cows would be highly contaminated. The radioactive fall-out would poison the vegetables we usually eat, and most of them would not grow again in our children's lifetime.

Mr. Harris got his information from an expert—who certainly furnished plenty of food for thought, at the expense of predicting that food for our stomachs would be too spoiled for human consumption.

* * *

HOPE FOR 1962

With this issue Volume 22 of the North Carolina Medical Journal comes to a close. To keep from ending the year on a sour note, the editors hasten to express the hope that reason will prevail against the idiocy of war, and that 1962 will see a greater willingness on the part of the world's people to heed the solemn warning: Love or perish.

Committees & Organizations

Public Relations Committee

REPORT ON THE A.M.A. INSTITUTE "MEDICINE IN ACTION"

Chicago, Illinois, August 31, and September 1, 1961

This annual two-day conference, formerly a public relations institute, has now been broadened in scope and has become the most interesting, concentrated, and fruitful session of its kind. Everyone who attends this meeting, including those who have attended many similar sessions in the past, returns home "loaded" with a satchel full of fresh ideas and inspiration. It is an outstanding accomplishment on the part of the A.M.A. and the participants. Herewith some comments on the program.

Two brilliant, exciting, and inspiring speakers were heard on the morning of August 31: the Rev. Robert Varley, Th.D., Rector of Salisbury Parish, Salisbury, Maryland, and a new member of the A.M.A. Speakers Bureau, delivered a forceful and thrilling address entitled "Are We Fighting A Battle We Can Win?" Among the vital points stressed by the Rev. Dr. Varley were: (1) Doctors must look to allies in *all* walks of life. "The medical world has always been the prime target for pseudo-social planners, not because you are rich and powerful . . . but rather because your training and professional insight represent the greatest threat to these idiot idealogists." (2) Stressing that the ranks of medicine must be united, he stated, "All need not agree in philosophy, but must agree in purpose."

The other "smash" talk of that morning was by Professor William DeMougeot of North Texas State University, Denton, Texas, who offered concise and beautifully analyzed tips in the use of the arguments against socialized medicine. He enumerated the arguments which have been and are being used and then discussed each one in detail, showing which are primarily emotional and which are primarily factual, and giving very useful suggestions on how to use them most effectively.

These two speakers have been invited to address the annual Officers' Conference for all county society officers and committee chairmen throughout North Carolina, scheduled for January 27, 1962, in Pinehurst. PLAN TO ATTEND.

The title of A.M.A. President Leonard Larson's talk was "A.M.A.'s Greatest Challenge," and this boiled down to be the apathy of so many physicians in the present situation, which requires active participation by *every* physician if we are to survive in our battle against the welfare state.

The other features of the program of the Institute were: a panel on how county and state medical societies implement A.M.A. programs; a panel on "How To Counter Criticism"; a panel on "Teamwork In Community Action"; and a radio-television roundtable moderated by the nationally known commentator, Alex Drier, which brought forth concise summaries of subjects such as the medical scholarship program, liaison with nurses, medical education, revamping of scientific publications, etc. A concluding and most interesting feature was an address by Professor Paul Peterson, University of Omaha, entitled "What's Right—And What's Wrong With AMA's Image?"

Those attending from North Carolina included President-elect John Kernodle of Burlington; Mr. W. N. Hillard of our State Society office in Raleigh; Dr. Marvin Lymberis, president-elect of the Mecklenburg County Medical Society, Charlotte; and Dr. David Welton, State Society public relations chairman, Charlotte.

David Welton, M.D.

* * *

Trust Study Committee

KEOGH BILL NOT PASSED

As most physicians are now aware, the Keogh Bill (HR 10) was not passed by the Senate during the first session of the eighty-seventh Congress which adjourned on September 27, 1961. This bill would have provided tax relief for self-employed persons contributing to private retirement pension plans somewhat comparable to the tax benefits enjoyed by officers and employees of corporations. The Senate Finance Committee made some alterations in the proposed bill and approved the altered bill by a vote of 13 to 3.

This is the closest this legislation has gotten toward enactment during the 12 years it has been before the Congress. It will not be necessary for it to originate again in the House of Representatives in 1962, as it may be taken up directly in the second session of this Eighty-seventh Congress.

At the present time the status of the legislation is that of awaiting consideration by a joint House-Senate committee which will work out a compromise of the changes made in the report of the Senate Finance Committee. The compromise version is expected to pass the House of Representatives and Senate sometime next year.

Apparently the only opposition to the Bill is coming from the Treasury Department, which claims that the Federal Government will lose 200 millions dollars in revenue unless self-employed individuals are continued to be denied the tax relief now given to corporation officers and employees.

Upon enactment of the "Self-employed Individuals Retirement Act" (HR 10), the Medical Society of the State of North Caroline will provide its members with the opportunity to participate in the North Carolina Medical Retirement Savings Plan, which will offer savings and advantage not available in individual plans.

JESSE CALDWELL, Chairman

W. B. SAUNDERS COMPANY features the following recent books in their full page advertisement appearing elsewhere in this issue:

Graham, Sotto and Paloucek—**Cancer of the Cervix** full and authoritative coverage of the diagnosis and management of cervical cancer—from Roswell Park Memorial Institute.

Hogan and Zimmerman — **Ophthalmic Pathology** an atlas and textbook on diagnosis of diseases of the eye and on the pathology of involved tissue.

Owen — **Hospital Administration** covers every aspect in the construction, organization and administration of today's hospitals.

Bulletin Board

COMING MEETINGS

North Carolina State Board of Medical Examiners meeting—Mid Pines Club, Southern Pines, January 12, 1962.

North Carolina Mental Health Association, Annual Meeting—Jack Tar Hotel, Durham, February 16-17, 1962.

Nineteenth Annual Watts Hospital Medical and Surgical Symposium—Watts Hospital and Jack Tar Hotel, Durham, February 23-24.

Gill Memorial Eye, Ear and Throat Hospital, Annual Congress on Ophthalmology and Otolaryngology—Roanoke, Virginia, April 2-6, 1962.

NEW MEMBERS OF THE STATE SOCIETY

The following physicians joined the Medical Society of the State of North Carolina during the month of October, 1961:

Dr. Matthew Fred John Yenney, Jr., 301 S. Grace Street, Rocky Mount; Dr. Amos Townsend Pagter, Jr., 100 Jervey Road, Tryon; Dr. George Victor Kokiko, Gaston Memorial Hospital, Gastonia; Dr. Graham Dougald Newton, 207 Hawthorne Lane, Charlotte; Dr. Robert Gardner Ellis, 21 E. Center Street, Lexington; Dr. Byron Atlee Nichol, 15 E. Center Street, Lexington; Dr. Edwin Lewis Auman, 330 Locke Street, High Point; Dr. Alfred Hamilton Garvey, 1311 N. Elm Street, Greensboro; Dr. Brooks Webster Gilmore, 342 N. Elm Street, Greensboro; Dr. Samuel Balfour Joyner, 1219 Magnolia Street, Greensboro; Dr. Vernon Wendell McFalls, 624 Quaker Lane, High Point; Dr. Oliver Fennell Roddey, Jr., 104 E. Northwood Street, Greensboro; Dr. Norman John Scheibling, Jefferson Standard Life Insurance Co., Greensboro; Dr. Paul Joseph Simel, 1311 N. Elm Street, Greensboro; Dr. Dwight Groome Davis, Jr., 3409 Cherry Lane, Raleigh; Dr. Richard Franklin Bowling, Shelby Medical Center, Shelby;

Dr. John Frederick Kappler, 131 W. Harper Avenue, Lenoir; Dr. Francis Joseph Kane, Jr., N. C. Memorial Hospital, Chapel Hill; Dr. Reed Porter Rice, N. C. Memorial Hospital, Chapel Hill; Dr. Everett Hoyle Schultz, Jr., N. C. Memorial Hospital Chapel Hill; Dr. Anthony Peter Slewka, Duke University Medical Center, Durham; Dr. Thomas Francis O'Brien, 2611 Buena Vista Road, Winston-Salem; Dr. Tom Phillip Coker, Bowman Gray School of Medicine, Winston-Salem; Dr. Harvey Lee Griffin, Jr., 213 Fayetteville Street, Asheboro.

NEWS NOTES FROM THE BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

Dr. Richard C. Proctor, chairman of the Department of Psychiatry, has been chosen president-elect of the Southern Psychiatric Association.

* * *

Dr. D. LeRoy Crandell, associate professor of anesthesiology, has been elected to the presidency of the North Carolina Society of Anesthesiologists. Dr. C. Max Drummond, instructor in anesthesiology, is secretary-treasurer of the organization.

* * *

At the annual meeting of the Bowman Gray Medical Alumni Association several papers relating to medico-legal problems were presented:

Dr. Luther R. Doffermyre, Dunn, "Narcotic Addiction Among Physicians"

Mr. Hoarce Cotton, Contributing Editor of **Medical Economics**, "The Business of Practicing Medicine"

Mr. Norman Block, Attorney, Greensboro, "The Role of Life Insurance in a Physician's Estate Plan"

Mr. Robert Clodfelter, Trust Officer of Wachovia Bank and Trust Company, was after-dinner speaker.

* * *

Recent appointments to the faculty include: Dr. James Francis Glenn, associate professor of Urology. Formerly a member of the Yale University School of Medicine faculty, Dr. Glenn completed his undergraduate education at the University. His internship and residency programs were completed at Peter Bent Brigham Hospital and at Duke Medical Center.

Dr. Charles D. Hendley has been named associate professor of pharmacology. He is a graduate of Swarthmore College, and earned the Ph.D. degree at Columbia University. Since 1953 he has been engaged in pharmaceutical research, and prior to that time held positions on the faculties of Ohio State University College of Medicine, South Dakoa School of Medicine, and Tulane University School of Medicine.

Dr. Ivan W. Davidson has assumed his duties as assistant professor of physiology and pharmacology. Since 1959 he has been associated with the research department of Union Carbide Chemicals. He received the Bachelor of Science degree at the University of Manitoba, and he earned the Master's and Ph.D. degrees at the University of Toronto.

Dr. Leonard A. Graham is instructor in pharmacology. He received the Bachelor of Science degree from Ohio State University, and the Master of Science and Ph.D. degrees at the University of Illinois College of Medicine. Dr. Graham has previously been engaged in research with the Armour Pharmaceutical Laboratories and the Department of Physiology of the University of Illinois College of Medicine.

To the part-time faculty have been added: Dr. Edwin L. Auman, assistant in clinical medicine; Dr. Edwin T. Bowen, Jr., assistant in Clinical pediatrics; and Dr. Robert E. Nolan instructor in clinical surgery.

NEWS NOTES FROM THE DUKE UNIVERSITY MEDICAL CENTER

Dr. Robert A. Gregg, formerly of Greensboro, has joined the Duke University Medical Center faculty as coordinator of rehabilitation.

Dr. Gregg will head the teaching program supported by the Office of Vocational Rehabilitation, U. S. Department of Health, Education and Welfare. His academic rank at Duke is associate professor of physical medicine.

Before joining the Duke faculty, Dr. Gregg served some two years as medical director of the Central Carolina Rehabilitation Hospital in Greensboro. He fills a position left vacant at Duke since the resignation of Dr. William D. deGravelles in 1958.

* * *

International tensions and the ever-present possibility of nuclear war have sparked a new program of fallout preparedness at Duke University.

A campus-wide Fallout Preparedness Committee is currently studying the best ways to protect the University community in case of a war involving nuclear weapons.

* * *

The Rev. Robert B. Claytor has been appointed to the newly established position of assistant chaplain to Duke Hospital.

He also will be associated with the Duke University Divinity School as chaplain supervisor for hospital clinical work involved in pastoral care courses.

* * *

An artifical hand currently being evaluated at the Duke University Medical Center promises new help for child amputees.

Developed in the Army Prosthetic Research Laboratory of Walter Reed Hospital, Washington, D. C., the hand is the first for young children that combines a life-like appearance with the ability to perform certain functions of a real hand.

Duke is one of 14 medical institutions in the United States now studying the hand to determine its effectiveness and find out what improvements are needed.

* * *

Dr. J. Graham Smith, Jr., dermatologist of Duke University Medical Center, was a speaker at the annual meeting of the Gerontological Society held in Pittsburgh last month. His report, based on research conducted with Drs. Eugene

A. Davidson, W. Mitchell Sams, Jr., and Richard D. Clark, dealt with chemical changes of the skin associated with aging and prolonged exposure to the sun.

Other Duke faculty members appearing on the program were Dr. Juanita Kreps of the Department of Economics and Dr. George L. Maddox, associate professor of sociology and of medical sociology.

Dr. Maddox's observations are based on a study of 251 elderly people, none of whom are in hospitals or homes for the aged.

News Notes from the University of North Carolina School of Medicine

Beginning in January, Tar Heel practicing physicians will be offered, without charge, three postgraduate training courses in psychiatry by the Department of Psychiatry of the University of North Carolina School of Medicine.

The courses, which will cover a period of three months, will meet weekly at the School of Medicine. They will include basic psychiatric principles applicable to the general practice of medicine, with emphasis on emotional and psychologic problems met by the practicing physician.

One course will be a lecture course and will be for an hour and a half a week. The second will be in the form of a seminar, also for an hour and a half, using case histories from the practice of the various attending physicians. The third course will involve supervised clinical work with psychiatric patients. The weekly sessions for this course will range from one half to one full day.

The faculty for the postgraduate courses will be drawn from the faculty of the Department of Psychiatry. It will include Dr. George C. Ham, Dr. Milton L. Miller, Dr. David R. Hawkins, Dr. John A. Ewing, and Dr. Charles R. Vernon.

Physicians who wish additional information on the courses are requested to write Dr. George C. Ham, U.N.C. School of Medicine.

* * *

Postgraduate medical courses sponsored by the Office of Continuation Education of the UNC School of Medicine and local medical societies got under way over station WUNC-FM on Tuesday, December 5.

The programs, each an hour long, will be given weekly for a 24-week period. A telephone arrangement will make it possible for the physicians to ask questions as well as listen to the lectures that will originate here at the School of Medicine.

The county medical societies taking part in the first 24-week program of instructions include Wilson, Wayne, Robeson, Lenoir, Lee, Chatham, Alamance, Granville, Rowan, and Guilford.

The cities in which the physicians will meet and where arrangements have been made for them to ask questions of the U.N.C. faculty members who will lecture are Wilson, Goldsboro, Lumberton, Kinston, Sanford, Siler City, Burlington, Oxford, Salisbury, and Greensboro.

While only those physicians in the organized groups will be able to take part in the question-and-answer part of the program, anyone with an FM radio who lives in the WUNC-FM area may tune in.

* * *

Dr. Henry T. Clark Jr., administrator of the University of North Carolina Division of Health Affairs, has been appointed a member of the General Clinical Research Center Committee of the National Institutes of Health.

This committee is one of several consultant groups being established by the surgeon general of the U. S. Public Health Service to assist the National Institutes of Health in reviewing applications for support of medical research.

* * *

Dr. D. Gordon Sharp, professor of biophysics, has been named to the Biophysical Sciences Training Committee of the Division of General Medical Sciences, National Institutes of Health. He and other members of the committee will review applications for training grants in the field of biophysical sciences.

* * *

A member of the U.N.C. faculty and an alumnus have been elected fellows of the American Academy of Pediatrics, according to Dr. E. H. Christopherson, executive director of the academy's international headquarters.

They are Dr. Floyd W. Denny Jr., professor and chairman, Department of Pediatrics of the U.N.C. School of Medicine, and Dr. William L. London, class of 1955, Durham.

North Carolina Board of Medical Examiners

The North Carolina Board of Medical Examiners will meet at the Mid Pines Club, Southern Pines, Friday, January 12, 1962, at which time applicants for license by endorsement will be interviewed.

North Carolina Pediatric Society

The following officers were elected to serve for 1962 at the annual meeting of the North Carolina Pediatric Society, held at Sedgefield Inn in Greensboro in November:

President—Dr. Weston M. Kelsey, Winston-Salem; president-elect—Dr. Angus M. McBryde, Durham; secretary-treasurer—Dr. F. A. Blount, Winston-Salem.

Serving on the executive committee will be Dr. Frank R. Reynolds, Wilmington; Dr. Fletcher L. Raiford, Hendersonville; and Dr. Katherine H. Anderson, Winston-Salem.

FORSTY-COUNTY MEDICAL SOCIETY

The Forsyth County Medical Society elected officers for 1962 at its regular monthly meeting held in Winston-Salem on November 14.

Dr. David Cayer, who has served as president-elect during the past year, will automatically succeed to the presidency, replacing Dr. James A. Harrill, retiring president. Other officers are Dr. Felda Hightower, president-elect; Dr. Carlton N. Adams, vice president; and Dr. Fred G. Pegg, secretary. Dr. Andrew J. Crutchfield was elected to the board of censors.

Fifty-year Club certificates, in recognition of 50 years of membership in local and state medical societies, were presented to Dr. James B. Whittington, Dr. Robert A. Moore, Dr. William P. Speas, and Dr. V. F. Couch.

Dr. James B. Field, of the clinical endocrinology branch of the National Institutes of Health, addressed the society on "Clinical Diagnosis of Hypoglycemia."

SPRING CONGRESS IN OPHTHALMOLOGY AND OTOLARYNGOLOGY

The Gill Memorial Eye, Ear and Throat Hospital at Roanoke, Virginia, will hold its thirty-fifth annual spring congress in ophthalmology and otolaryngology and allied specialties April 2-6, 1962. Guest speakers will be drawn from a wide area within the United States, and will include one physician, Dr. P. D. Trevor-Roper, from London, England.

For further information write the Superintendent, P. O. Box 1789, Roanoke, Virginia.

SOUTH CAROLINA BOARD OF MEDICAL EXAMINERS

The State Board of Medical Examiners of South Carolina now has an Official Directory containing a list of all the licensed physicians within the state and the Medical Practice Laws of the State of South Carolina. The list gives the physician's license number, name, type of practice, and the address.

The directory is available at $2.00 per copy. Please make remittance to the State Board of Medical Examiners of South Carolina, N. B. Heyward, Executive Secretary, 1329 Blanding Street, Columbia, South Carolina.

AMERICAN COLLEGE OF GASTROENTEROLOGY

Dr. Edward J. Krol of Chicago, was chosen as president-elect of the American College of Gastroenterology, at the annual meeting of the College held on October 22 in Cleveland, Ohio.

Dr. Louis Ochs, Jr. of New Orleans, Louisana,

1 2 3 4
clinical studies repeat...

ARLIDIN IMPROVES HEARING[1]
ARLIDIN IMPROVES HEARING[2]
ARLIDIN IMPROVES HEARING[3]
ARLIDIN IMPROVES HEARING[4]

Arlidin is available in 6 mg. scored tablets,
and 5 mg. per cc. parenteral solution.
See PDR for packaging.
Protected by U.S. Patent Numbers: 2,661,372 and 2,661,373.

1 "significant hearing improvement" occurred with Arlidin in 32 of 75 patients with recent onset hearing impairment due to labyrinthine artery ischemia.

Rubin, W. and Anderson, J. R.: Angiology 9:256, 1958.

3 Arlidin "appears to be one of the most satisfactory [vasodilators], having the advantages of minimal side effects, being well tolerated and possessing a sustained action" in improving circulation of the inner ear.

Seymour, J. C.: Laryngology & Otology 74:133, 1960.

selected president-elect in Philadelphia in 1960, assumed the presidency of the College, succeeding Dr. Henry Baker of Boston.

American Board of Obstetrics and Gynecology

The next scheduled examinations (Part II), oral and clinical, for all candidates, will be conducted at the Edgewater Beach Hotel, Chicago, Illinois, by the entire Board from April 9 through 14, 1962. Formal notice of the exact time of each candidate's examination will be sent him in advance of the examination dates.

Candidates participating in the Part I Examination will be notified of their eligibility for the Part II Examinations as soon as possible.

Current Bulletins of the American Board of Obstetrics and Gynecology outlining the requirements for application may be obtained by writing to the Secretary; Robert L. Faulkner, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

National Medical Foundation for Eye Care

The National Medical Foundation for Eye Care has announced the names of 1961-1962 officers elected recently at the Foundation's annual meeting in Chicago.

Re-elected president was Dr. Ralph O. Rychener of Memphis, Tennessee, Dr. William B. Clark of New Orleans, Louisiana, was named vice president, and Dr. Charles E. Jaeckle of Defiance, Ohio, was re-elected secretary-treasurer.

Dr. S. D. McPherson, Jr., of Durham, North Carolina, is a member of the board of trustees.

CLASSIFIED ADVERTISEMENTS

POSITION OPEN: Nurse Anesthetist, CRNA preferred, 70 bed hospital in Northwestern Montana. Excellent opportunity, liberal benefits. Salary open. Contact Administrator; Kennedy Deaconess Hospital; Havre, Montana.

WANTED: Male psychiatrist under age of 50 years. Board certified or Board eligible to head established private psychiatric clinic in city 75,000. Graduate American School of Medicine. Salary: $18,000—$20,000, depending upon qualifications and experience, plus liberal fringe benefits. Incentive plan permits additional earnings of $5,000 annually. Address Box 790, Raleigh, N. C., in care of this Journal.

NEEDED: General practitioner with major surgical experience, including at least two (2) years formal surgical postgraduate training. The medical staff of new 50-bed fully accredited general hospital desires permanent location of above type physician. Full cooperation is pledged and association is possible if desired.

2

vascular insufficiency of the labyrinth is an important etiologic factor in sudden perceptive deafness . . . "vasodilators [Arlidin] are of considerable value."

Wilmot, T. J. and Saymour, J. C.: Lancet 1:1098, 1960.

4

early cases of sudden perceptive deafness should be treated by immediate stellate block "supplemented by the most effective vasodilator drug [Arlidin] . . . energetic measures to retain blood supply to the inner ear are imperative."

Wilmot, T. J.: J. Laryngology & Otology 73:466, 1959.

in impaired hearing, tinnitus, vertigo . . .

when due to ischemia of the inner ear . . .

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brand of nylidrin hydrochloride N.N.D.

Clinical benefit in approximately 50% of cases of recent onset hearing loss treated with adequate vasodilator and other supportive therapy is also reported by Sheehy.

Sheehy, J. L.: Laryngoscope 70:885, 1960.

CAUTION: Like any effective peripheral vasodilator, Arlidin should be used with caution in the presence of recent myocardial lesions, severe angina pectoris and thyrotoxicosis. There are no known contraindications to its use. Complete detailed literature available to physicians.

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Book Reviews

Pathology. Edited by W. A. D. Anderson, M.D., Edition 4. 1,389 pages. Price, $18.00. St. Louis: The C. V. Mosby Company, 1961.

The fourth edition of Anderson's text on pathology includes the contributions of 35 authors. The format remains unchanged, although there are three pages less and 87 more illustrations. There is a new chapter on hypersensitivity states and another on mesenchymal tumors of soft tissues. Many of the conditions omitted from the index in earlier editions now have an entry.

From the standpoint of completeness, this is probably the best single-volume text on pathology now available. The quality of individual contributions is uneven, and at times one might wish for a more firm editorial hand. In general, however, the work is adequate.

As Dr. Anderson states in his preface, the undergraduate student will find the work useful with the teacher as guide. In the years beyond medical school it should be particularly valuable as a reference book when the physician lacks the time or the opportunity to consult the current literature, yet desires to know the pathologic features of a given condition.

Proceedings: Thirteenth International Congress on Occupational Health. New York, Book Craftsmen Associates, Inc., 1961. 1005 pages. Price $10.00.

The Thirteenth International Congress on Occupational Health was held in New York City July 25-29, 1960. Its proceedings are published in a huge volume of more than one thousand pages.

Twenty four pages were required to list the contributions—nearly three hundred of them—to this volume. Most of the papers are in English, but some are in French, Italian, Spanish, and German; none were noted in the Russian language.

After introductory reports and addresses, papers read to the Congress were grouped under the following main headings:

1. Administrative practices.
2. Surgical practices.
3. Medical practices.
4. Education and training.
5. Environmental hygiene.
6. Social and legal aspects.
7. The influence of environmental factors on health.
8. Work physiology and psychology.

The papers that seemed of particular interest to this reviewer were as follows:

"Nursing Administration in Hospital Employee Health Services."
"The Surgeon's Opinion: A Major Factor in the Prolongation of Uncomplicated Surgical Convalescence."
"A Practical Approach to the Tetanus Problem."
"The Nurse Consultant."
"Congestive Heart Failure and Employment."
"Evaluation of a Treatment Program for Alcoholics in Industry."
"The Expensive Back."
"Suicide During Productive Years—an Industrial Problem" (by Drs. Mac Roy Gasque and Carl S. Plumb, from the Olin Mathieson Chemical Corporation, Pisgah Forest, N. C.)
"A Study of the Influence of Immunization on the Clinical Course of Asian Influenza in an Industrial Community."
"An Analysis of Sickness Absenteeism in a Bank."
"Occupational Toxic Anemia."
"Management Health Examination Program in General Motors."
"The Work Performance of Epileptics in Industry."
"Health Education of the Workers."
"The Cardiac Case in Workmen's Compensation."
"Court Cases of Alleged Occupational Dermatitis."
"Physiological Aspects of Shiftwork."
"Measurement of Human Reaction to Hardness of Floor Covering."
"Capitalizing on Stress."
"Medical Aspects of Motor Vehicles Accident Prevention."

Mr. J. T. Barnes states that this volume is in the headquarters office of the State Medical Society, where it is available for reference.

The Fourteenth International Congress on Occupational Health will be held in Madrid in 1963.

The Month in Washington

The Public Health Service said that radioactive fallout levels resulting in the United States up until early November from the new series of Soviet nuclear explosions "do not warrant undue public concern" nor initiation of any special public health action.

The federal agency said that the prevailing levels were not high enough for the public to be concerned about the safety of milk and other foodstuffs, but added that "continuous, intensive surveillance" by federal, state, and local governments was justified.

In a special statement issued after a two-day conference of government and private radiation experts, the PHS pointed out that "very little is known about the effects on

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Abbreviations

C—Correspondence
C&O—Committees and Organizations
PCC—Poison Control Center
PM—President's Message

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The Month in Washington

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animals or humans of very low but prolonged exposures" from either natural background radiation or fallout from nuclear tests.

"The consensus of scientific opinion is that the most prudent course is to assume there is no level of radiation exposure below which one can be absolutely certain that harmful effects may not occur to at least a few individuals when sufficiently large numbers of people are involved," the PHS said. "This is known as the 'non-threshold' concept."

This concept is the basis for U. S. policies and programs for assessment of radiation hazards and for control measures designed to limit exposures of the population, the PHS said, and added: "When this non-threshold concept is applied to present radiation exposure levels being experienced in the U. S. from all sources, including fallout, the following assessment can be made:

> "The extra radiation caused by the Soviet tests will add to the risk of genetic effects in succeeding generations, and possibly to the risk of health damage to some people in the United States. It is not possible to determine how extensive these ill effects will be—nor how many people will be affected. At present radiation leves, and even at somewhat higher levels, the additional risk is slight and very few people will be affected. Nevertheless, if fallout increased substantially, or remained high for a long time, it would become far more important as a potential health hazard in this country and throughout the world.

"It is the obligation of our Federal and State governments to undertake all possible measures to assess accurately the public health significance of the present fallout situation, and to prepare for actions to safeguard the public health if these become necessary."

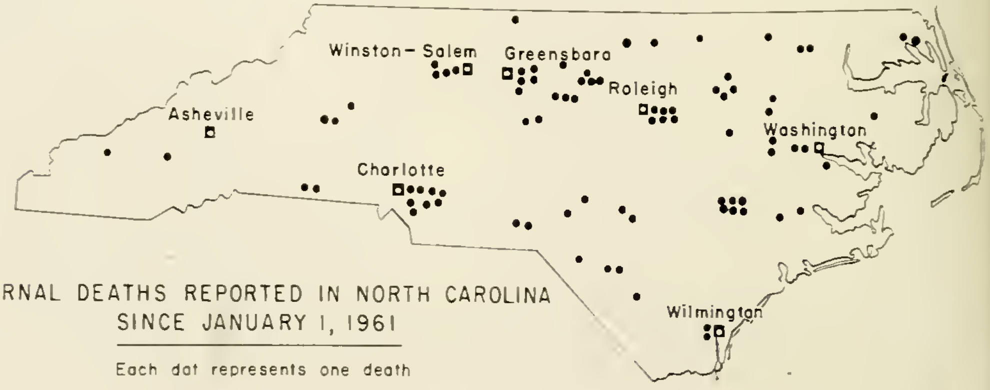
Federal officials said radioactive fallout on the United States will increase next February, March, April and May, when the late winter and spring rains wash to earth the remainder of the fallout from the Soviet nuclear tests, but it is not expected to reach a dangerous level. President Kennedy said any U. S. nuclear tests in the atmosphere would be designed to hold radioactive fallout to an absolute minimum.

The PHS said that the nation's health authorities are giving careful consideration to the possible situations that might require various corrective actions. "It is evident that an important element of health protection is continuous surveillance and analysis," the PHS said.

Winthrop Completes Film On Physician Drug Addicts

Winthrop Laboratories has completed a new motion picture on the subject of drug addiction, and is making prints available for showing to professional groups.

Titled "Face of an Addict," the film was produced with the cooperation of the Division of Narcotics Control and the Mental Health Division of Canada's Department of National Health and Welfare. It was filmed in Canada by Crawley Films, with Dr. Martin Lasersohn, vice president of Winthrop, serving as technical advisor.



MATERNAL DEATHS REPORTED IN NORTH CAROLINA SINCE JANUARY 1, 1961

Each dot represents one death